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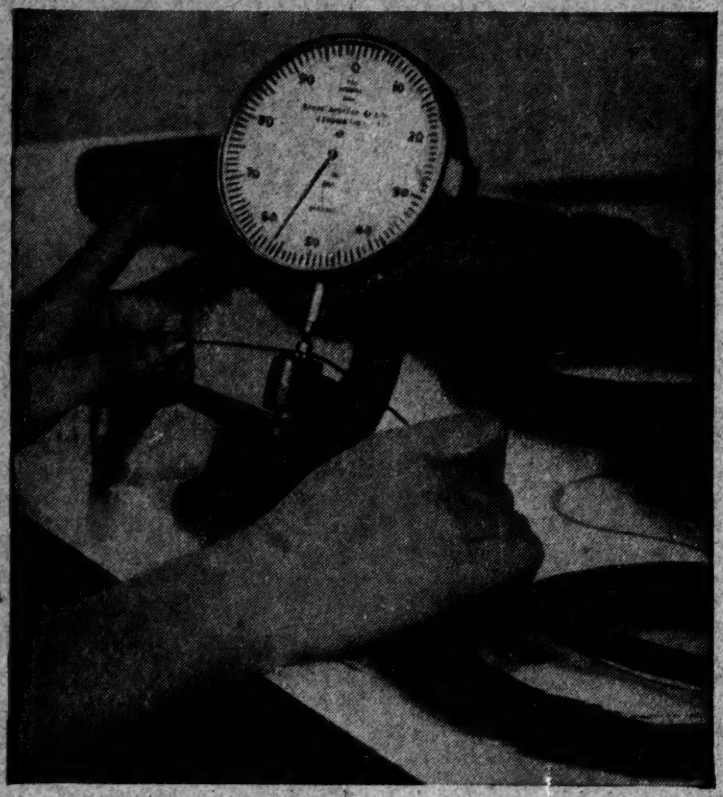

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THE
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VOL. XXXII JANUARY, 1945 No. 127

WOUNDS OF THE COLON

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THIS review of wounds of the colon, though not complete owing to the fact that most of the data were obtained from reports at Base Hospitals, will, it is hoped, be useful and of interest, since not only has the average war surgeon a restricted experience of colon wounds, but he is often unable to follow up all his cases in detail.

The improved results of war surgery of the abdomen are, in the main, the outcome of all those factors which, in the fifteen years or so before the outbreak of war, were producing better results in civil surgery. It is in the field of pre-operative and post-operative treatment that the most important advances in abdominal surgery have been made in recent times. Surgeons in the Middle East were privileged to be amongst the first to apply these advances in surgery to the treatment of wounds in battle.

The time-lag factor in wounds of the abdomen, though important, must not overshadow the sober need for skilled post-operative nursing. A lone Field Surgical Unit (F.S.U.) working with a Field Ambulance, though it can deal with a proportion of wounds of the first urgency—hæmorrhage, sucking chest wounds, and abdominal wounds—is of little avail if the latter succumb from lack of skilled after-care. It is a fact that abdominal cases travel badly, but it is not so much the ill effects of travelling that matter, as the difficulty of proper pre- and post-operative care during transit. A forward surgeon who operates on abdominal wounds must be so placed that he can retain his patients for at least a week or preferably 10 to 14 days, and have at hand all the necessities for thorough treatment and nursing. The surgeon must not be regarded as a travelling saviour of lives, but part of a team which can give the best treatment to the largest number of wounded, an essential part of this team being a Field Transfusion Unit (F.T.U.). He must be available to give cases of internal hæmorrhage a chance of survival, though severe internal hæmorrhage is usually beyond surgical aid. Treatment of wounds of the abdomen can wait, when necessary, for a few hours, since gastro-intestinal contents do not, as a rule, pour out into the general peritoneal cavity. Although it is undesirable as a general rule, in exceptional circumstances, such as commando raids or during the initial period of a sea or airborne landing, or when the military situation demands it, F.S.U.'s and F.T.U. may work with a Field Ambulance, since rapid evacuation of wounded may be impossible.

It has been found that two F.S.U.'s and one F.T.U., attached to a Casualty Clearing Station (C.C.S.), a light section of the C.C.S., or a Field Dressing Station, form a good working arrangement, since it minimizes pre-operative delay. The F.T.U. not only prepares and helps to select cases for operation, but also assists in the post-operative treatment, especially the parenteral administration of fluids, etc. Grouping of C.C.S.'s with their attached F.S.U.'s and F T.U.'s to form an Operating Centre is sometimes desirable. Reserve F.S.U.'s may be held in readiness and moved rapidly, with or without their basic operating equipment, to relieve or to assist overworked teams.

In this war the surgeon is probably seeing and operating on cases which, in the last war, died before reaching skilled aid; almost hopeless cases have been operated upon. An increased operability rate, in spite of an increased mortality, will, up to a point, save more lives, but the optimum limit and the limit of optimism may easily be passed unless the young and fearless surgeon is placed not only in the right tactical, but in the correct surgical, position. Front-page drama in abdominal surgery does not pay—the handicaps of working without a Transfusion Unit, a skilled anæsthetist, a good light, an adjustable operating table, and adequate arrangements for nursing and after-care, are too great for even the most enthusiastic surgeon.

It is with sincere admiration and homage that I add my tribute to the skill, courage, perseverance, and judgement of the forward surgeons in whose hands the badly-wounded soldier is placed.

It is unwise to draw too many conclusions from study of wounded at the Base, since the

forward surgeon alone knows of his failures and disappointments; the survivors tell only of the triumphs. It will be realized that a primary mortality figure cannot be given, since most of the case reports analysed in this study are of those who survived their wounds. Major-General Ogilvie, in his masterly and frank review of "Abdominal Wounds in the Western Desert", showed the following mortality rates :—

All cases (complicated and uncomplicated): Colon, 51·5 per cent; colon and small intestine, 62·5 per cent (cf. 74 per cent in last war).

Uncomplictaed cases: Colon, 43·9 per cent; colon and small intestine, 58·6 per cent.

Uncomplicated cases operated upon within 12 hours: Colon, 40 per cent; colon and small intestine, 55·5 per cent.

From the following analysis of 122 cases of wounds of the colon, 109 penetrating and 13 non-penetrating, there emerge, however, certain interesting facts.

Frequency of Site of Injury.—In 120 soldiers with 126 injuries of the colon, it was found that the right colon (cæcum, ascending colon, and hepatic flexure), the transverse colon, and the left colon (splenic flexure, descending colon, and sigmoid colon) were injured in about equal frequency. The transverse colon, however, was twice as often the site of damage when compared with each anatomical portion of the large intestine (*Fig.* 269, A). The same relative proportions were found when survivors alone were considered (*Fig.* 269, B).

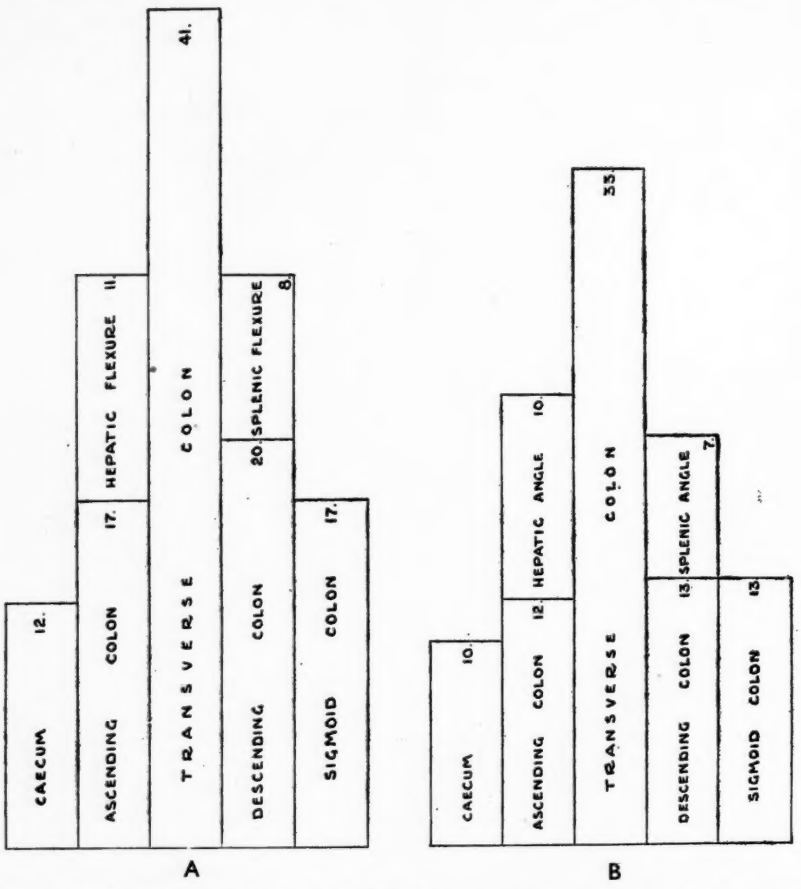

FIG. 269.—A, Relative sites of colon injuries (126 colon wounds in 120 patients). B, Relative sites of colon injuries in survivors (98 lesions in 94 patients).

It must be remembered, however, that there is no information, in this study, of the deaths from wounds of the colon on the battle fields or in the forward units.

Site of Entry Wound in Relation to Portion of Colon Injured.—In 106 wounds of the colon in which the position of the wound of entry was known, there was approximately an equal number of right, left, and transverse colon lesions, but a difference in the frequency of the site of the entry wounds was apparent (*Fig.* 270, A).

34 wounds right colon—abdominal entry wound in 15 instances (44 per cent).
35 wounds transverse colon—abdominal entry wound in 25 instances (70 per cent).
37 wounds left colon—abdominal entry wound in 13 instances (35 per cent).

A more detailed analysis of the position of the entry wound in relation to the site of colon

damaged is shown (*Fig.* 270, B). The relation of the wound of entry to the wound in the colon in survivors is shown (*Fig.* 271, A, B).

It will be seen that, whilst the cæcum and transverse colon were most frequently injured from the front, the ascending colon with the

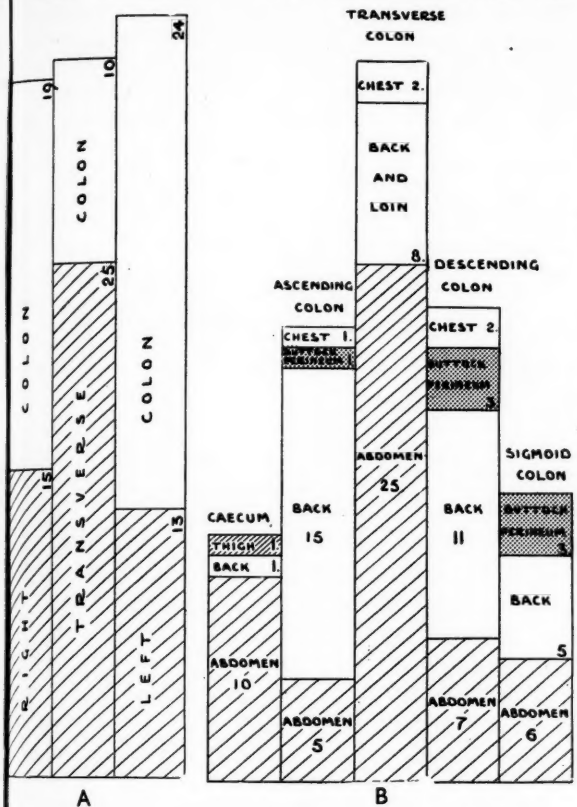

FIG. 270.—A, Analysis of 106 wounds of the colon showing relative frequency of entry wounds in anterior abdominal wall compared with entry wounds elsewhere. Anterior abdominal entry wounds shaded. B, Detailed analysis of wounds shown in A.

hepatic angle, and the descending colon including the splenic angle, were usually injured from the back. The sigmoid colon sustained damage from missiles entering either anteriorly, posteriorly, or from below. It is noteworthy that the fixed ascending and descending colon were not as commonly injured from the front as the other portions of the colon.

Wounds of the lower quadrants of the abdomen appear to have involved the colon twice as frequently as those in the upper half (*Table A*).

Relation of Time-lag, Recovery, and Site of Colonic Injury.—The time-lag was known in 63 cases surviving for 28 days or longer, but 7 of these had non-penetrating wounds. Accordingly, only 56 cases are available for this analysis (*Fig.* 272).

It will be noted that 17 cases recovered after a time-lag of over 12 hours (over one-third); 8 cases recovered after over 18 hours (one-seventh); 5 cases recovered after a pre-operative delay of 24 hours (one-eleventh).

In late cases a surgeon may be rewarded more often than is generally realized.

From a study of *Fig.* 272, it will be seen that among cases operated upon within 6 hours there is a preponderance of those with injury to the transverse colon, whereas in the second 6-hour period there is a big fall in the number of survivors following injury of this segment.

Associated intra-abdominal injuries complicating wounds of the colon are most frequent in the case of the transverse colon (63 per cent) (*see Tables C* and *D*).

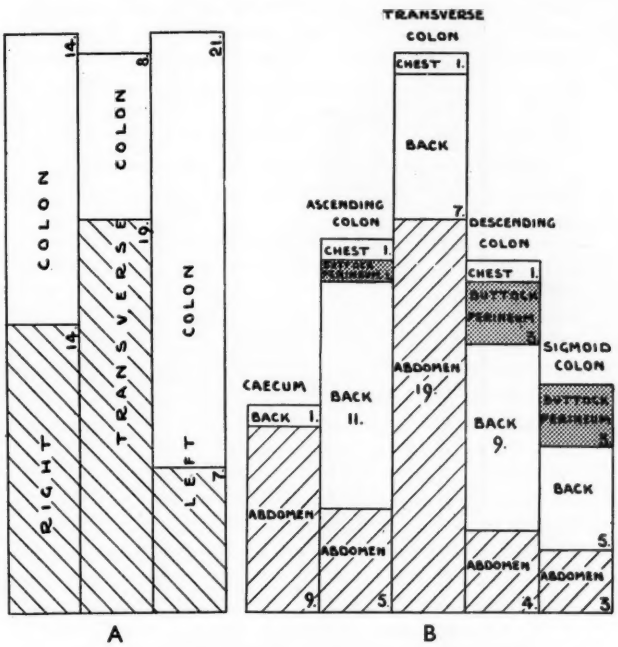

FIG. 271.—A, Analysis of 83 wounds of the colon in survivors. Those with anterior abdominal entry wounds are shaded. B, Detailed analysis of wounds shown in A.

Table A.—SITE OF ABDOMINAL ENTRY WOUNDS IN QUADRANTS
(38 *cases*; 28 *survivors*)

RIGHT UPPER QUADRANT	LEFT UPPER QUADRANT
7 {All wounds of transverse colon	6 {3 Transverse colon 3 Splenic angle
[Survivors— 7 {All wounds of transverse colon]	[Survivors— 3 {1 Transverse colon 2 Splenic angle]
RIGHT LOWER QUADRANT	**LEFT LOWER QUADRANT**
11 {7 Cæcum 1 Ascending colon 3 Transverse colon	14 {6 Transverse colon 4 Descending colon 3 Sigmoid colon 1 Cæcum
[Survivors— 9 {6 Cæcum 3 Transverse colon]	[Survivors— 9 {5 Transverse colon 2 Descending colon 1 Sigmoid colon 1 Cæcum]

The number of survivors is approximately equal for wounds of each segment of the colon (*Fig.* 269, B); of 98 wounds of the colon in 94 survivors, there are 32 right colon lesions, 33 transverse colon lesions, and 33 left colon lesions.

Bearing these facts in mind, the sudden fall in the number of cases of injury of the transverse colon surviving after the first 6-hour period is probably due to the increased risk of severe hæmorrhage from the wide expanse of vascular mesentery, mesocolon, omentum, or from injury

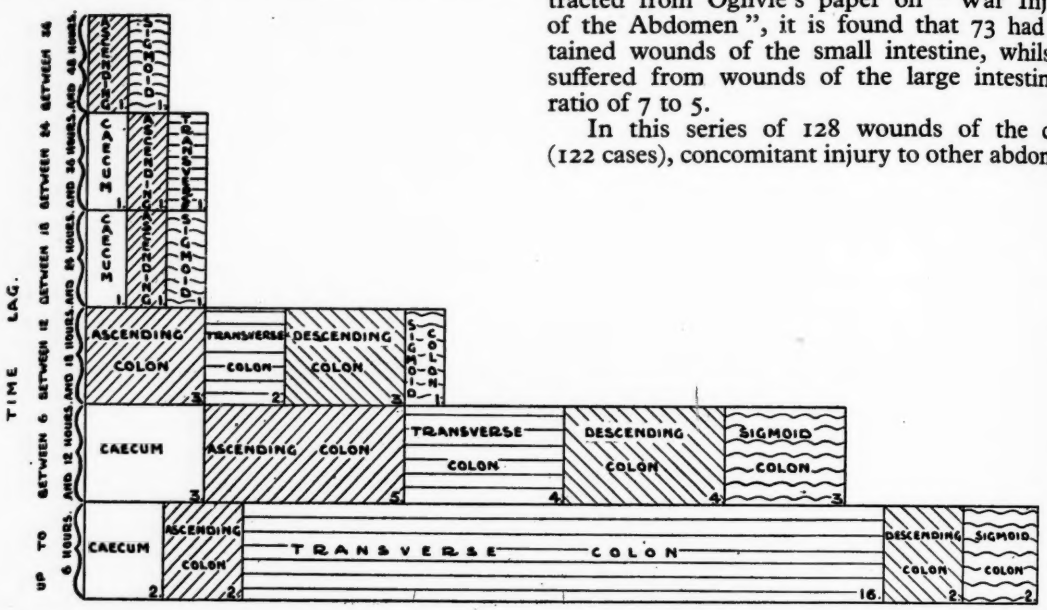

FIG. 272.—Sixty penetrating wounds of the colon in 56 survivors (over 28 days) showing time lag and site of lesion. There were 4 cases with wounds of 2 portions of the colon. There were 4 transverse colons with associated wounds in the descending colon 2, ascending colon 1, and sigmoid colon 1; all were cases within 12-hours time lag.

to other abdominal organs. Unless the cases reach the surgeon quickly enough, hæmorrhage will rapidly prove fatal. Once hæmorrhage is controlled, there are, in general, few technical difficulties attending the treatment of a wound in this portion of the colon, owing to its mobility.

One might have expected that wounds of the right and transverse portions of the colon would be less lethal than the remainder, since not only are the organisms less virulent, but the cæcum and transverse colon can be exteriorized with the minimum of interference, whereas on the left side, apart from the sigmoid loop, mobilization is difficult. The better blood-supply of the proximal colon increases the risk of severe hæmorrhage, and this, together with the greater frequency of other abdominal injury, balances the increased chances of infection experienced in the surgery of the left colon.

It is difficult at this stage to obtain a large enough series of wounds of the colon sufficiently recorded to form a definite opinion as to whether a soldier surviving the first 12 hours is more likely to live with a wound of the proximal than with one of the distal colon. However, in the small group of 17 cases surviving after a time-lag of 12 hours, 11 were in the right and transverse colon, and of the remaining 6, 3 were in the mobile sigmoid (*Fig.* 272).

Frequency of Associated Wounds of Other Abdominal Organs.—It is well known that the small intestine is the abdominal organ most frequently involved in penetrating injuries of the abdomen, but wounds of the colon occur not much less frequently. In 205 penetrating wounds involving the abdominal viscera, extracted from Ogilvie's paper on "War Injuries of the Abdomen", it is found that 73 had sustained wounds of the small intestine, whilst 53 suffered from wounds of the large intestine—a ratio of 7 to 5.

In this series of 128 wounds of the colon (122 cases), concomitant injury to other abdominal organs occurred in 42 per cent. The organ most frequently injured in addition to the large gut was the small intestine; this occurred in 31 per cent; the small intestine and colon alone were involved in 25 per cent. In 9 per cent of

Table B.—INCIDENCE OF ASSOCIATED INJURY OF OTHER ABDOMINAL ORGANS WITH LESION OF THE COLON

SITE OF INJURY	128 COLON LESIONS IN 122 PATIENTS (ALL CASES)	99 COLON LESIONS IN 95 SURVIVORS AFTER 28 DAYS
Colon with lesions of other abdominal organs	54 (42 per cent)	39 (40 per cent)
Colon with lesions of small intestine with or without other organs	40 (31 per cent)	29 (29 per cent)
Colon with lesions of small intestine only	32 (25 per cent)	24 (24 per cent)
Colon with more than one other lesion of an abdominal organ	12 (9 per cent)	6 (6 per cent)

the cases, more than one organ in addition to the colon was injured (*Table B*). The incidence of associated abdominal injury amongst those who survived wounds of the colon is also shown (*Table B*).

The frequency of injury to other abdominal viscera in terms of the particular segment of the colon damaged is shown in *Tables C* and *D*. It will be seen that associated intra-abdominal injury occurred about twice as frequently with injuries of the transverse colon as with any other portion of the colon.

Table C.—INCIDENCE OF ASSOCIATED INTRA-ABDOMINAL INJURIES WITH EACH SEGMENT OF THE COLON
(126 *colon lesions*; 120 *patients*)

SEGMENT OF COLON INJURED	NUMBER OF LESIONS IN OTHER VISCERA		
Transverse colon 41 lesions	26 (63 per cent)	Small gut	15
		Small gut and liver	1
		Small gut and stomach	2
		Small gut, liver, and stomach	1
		Small gut and kidney	2
		Liver	3
		Liver and spleen	1
		Stomach	1
Right Colon 40 lesions	11 (27·5 per cent)	Cæcum — Small gut	3
		Ascending — Small gut	2
		Ascending — Liver and kidney	1
		Hepatic angle — Liver	4
		Hepatic angle — Kidney	1
Left colon 45 lesions	16 (35·5 per cent)	Splenic angle — Small gut	1
		Splenic angle — Small gut, kidney, liver, and spleen	1
		Descending — Small gut	2
		Descending — Stomach and spleen	1
		Descending — Liver and kidney	1
		Sigmoid — Small gut	8
		Sigmoid — Small gut and bladder	1
		Sigmoid — Bladder	1

Table D.—INCIDENCE OF ASSOCIATED INTRA-ABDOMINAL INJURIES WITH EACH SEGMENT OF THE COLON
(*Survivors only*: 98 *colon lesions*; 94 *patients*)

SEGMENT OF COLON INJURED	NUMBER OF LESIONS IN OTHER VISCERA		
Transverse colon 33 lesions	21 (63·6 per cent)	Small gut	13
		Small gut and liver	1
		Small gut, liver, and stomach	1
		Small gut and kidney	1
		Liver	3
		Liver and spleen	1
		Stomach	1
Right colon 32 lesions	8 (25 per cent)	Cæcum — Small gut	3
		Ascending — Small gut	1
		Hepatic angle — Liver	3
		Hepatic angle — Kidney	1
Left colon 33 lesions	10 (30 per cent)	Splenic angle — Small gut	1
		Splenic angle — Small gut, kidney, and spleen	1
		Descending — Small gut	1
		Sigmoid — Small gut	5
		Sigmoid — Small gut and bladder	1
		Sigmoid — Bladder	1

Of the solid viscera, the liver was the one most frequently involved, and the associated colonic injury was usually in the hepatic angle or transverse portions.

In this series, the colon was damaged in more than one segment on six occasions; 2 of these cases died.

It is of interest that fractures of the ilium occurred seven times with injury of the left colon, twice with injury of the right colon, and once with injury of the transverse colon.

Resuscitation.—The work of the Transfusion Units and the regular supply of blood, plasma, and other intravenous fluids were monuments to organization, skill, and thoroughness. Blood was always available and was delivered as regularly as the peace-time milk supply—only over scattered areas of the desert. As the army moved westwards after the turning point at El Alamein, the "Sign of the Red Vampire" made its appearance soon after the tanks rumbled into a fallen town.

The resuscitation service had two tasks: the first to make the wounded fit for operation, and the second to maintain fluid and salt balance during convalescence in men who were often dehydrated and deficient in sodium chloride from sweating, etc. Close co-operation between the surgeon, transfusion officer, and anæsthetist was essential for the correct selection of the case and of the time for operation.

Of 28 cases in this series in which there was marked shock requiring pre-operative blood transfusion, 25 had intraperitoneal or an extensive extraperitoneal hæmorrhage. In the remaining 3 there were other injuries which produced shock. It may be said that in uncomplicated penetrating wounds of the abdomen, shock is synonymous with internal hæmorrhage.

The wounded die in the first few hours from hæmorrhage and shock. Rapid restoration of the blood-volume, or, when bleeding is active, *maintenance* of the circulation until hæmostasis can be secured, is only possible when unlimited quantities of blood or plasma are available: such supplies were always at hand.

At the second battle of El Alamein 9 per cent of the casualties required transfusion, whilst at the battle of Mareth transfusion was neeessary in 18 per cent—this was probably due to the greater incidence of shock-producing mine wounds. In this series the average amount of intravenous blood or plasma was 3·8 pints per patient, though much larger quantities are recorded. The largest amount in this series was 7 pints.

Plasma transfusion by Regimental Medical Officers was often possible well forward, especially in armoured formations or when there was a delay in evacuation. The difficulty of maintaining blood-volume during evacuation was overcome to some extent by arranging continuation of transfusion in the ambulance or even in aircraft.

Operative Technique.—As a general rule complete exploration of the abdomen can only be carried out through a median vertical incision. When damage to the fixed portion of the colon is suspected or found, much time is saved by making a separate lateral oblique or transverse muscle-cutting incision; examination, mobilization, and satisfactory exteriorization will thus be made easier.

Division of the rectus abdominis muscle above the umbilicus may be required, but, with an oblique or transverse incision, complete division of the outer edge of the rectus sheath will allow the edges of the incision to be retracted to a much greater extent.

Transverse or oblique incisions, when too near the costal margin or the iliac crest, are not only difficult to suture, but closure of a colostomy under these circumstances will be more troublesome.

The fixed portions of the colon appear to be more frequently injured from the back, and, therefore, with a penetrating wound of the flank, the wound should first be trimmed and then explored. The wound may be enlarged obliquely forwards, the abdomen opened, and the peritoneal aspect of the colon examined. Leakage from an extraperitoneal perforation is temporarily controlled, before the peritoneal cavity is opened, by means of a swab held with sponge-holding forceps.

The extent of the abdominal exploration will depend upon the pre-operative physical signs, the position of the foreign body as shown by X-ray examination, and upon the degree of hæmorrhage or soiling. In many of the cases reviewed, an oblique incision only had been used; in each case, of course, the advisability of making a further median exploratory incision must be decided.

When the abdomen has been opened through a right oblique incision, discovery of bruising or injury to the jejunum, its mesentery, or left portion of the omentum will demand exploration of the left side of the abdomen and/or pelvis. Evidence of injury to the terminal ileum, its mesentery, or the right omentum, when seen through a left oblique incision, will indicate the need for examination of the right side of the abdomen and pelvis. An additional midline incision may, therefore, be necessary to complete the examination of the abdominal contents.

The insertion of a dry swab on a holder into the pelvis, though apt to mislead, may sometimes be justifiably used as a means of assessing the probability of intra-abdominal injury in a very ill patient. The escape of gas or a fæcal odour is an important observation.

Decision on the best line of treatment must not be made before complete assessment of all the damage: with multiple lesions of the gut each may be marked with forceps for rapid identification. Localized paresis and distension of a loop of small intestine are usually associated with injury to this portion of the gut. Exploration must be quick and methodical; hæmorrhage demands examination of the solid viscera first and must be controlled before further exploration.

When the wound of entry is far back in the thickness of the loin, if its exploration does not quickly give a clue to the extent of the injury, the abdomen should be opened through a midline incision.

Treatment of the Injured Colon.—Though, in the past, colostomy for wounds of the colon has shown a higher mortality rate than suture, it must be remembered that this method had usually been employed in cases with extensive damage to the bowel. Exteriorization of the damaged portion of the colon, when possible, is the safest procedure. A temporary fæcal fistula is of little consequence if life is saved. In the mobile segments of the large intestine this is easy and may be quickly accomplished. If circumstances permit, fixed portions should be mobilized and brought outside the peritoneal cavity. Though it is better to bring the gut out through the skin, in urgent cases with a short loop, extraperitonealization may be achieved by suturing the injured colon to the parietal peritoneum; the abdominal wall should not be completedly sutured.

When the condition of the patient is such that the risk of mobilization is too great or is impossible, closure of the perforation, using two layers of sutures, together with its complete exclusion by a proximal colostomy is the next best method. The colostomy must have a good spur and should be established as near the lesion as possible; the sutured area should be drained thoroughly. Where the lower third of the pelvic colon is involved, adequate exposure and suture are difficult, and drainage and proximal colostomy alone may have to suffice. The value of local sulphadiazine, especially under these circumstances, appears to warrant its continued use. At the end of the operation 10 g. of sulphadiazine in suspension are injected through a catheter placed down to the infected area (*see below*).

A grossly bruised and devitalized area of bowel should also be treated as a penetration, since not only is there sometimes difficulty in outruling a perforation, especially when extraperitoneal, but there is also danger of delayed perforation resulting from necrosis of the bowel wall due to thrombosis.

Extraperitoneal perforation, especially of the fixed portions of the colon, may easily be overlooked. Mobilization of the colon for inspection of its extraperitoneal aspect is indicated when there is extraperitoneal bruising or gas, or evidence of injury in the immediate vicinity. Such a lesion in the fixed portion of the colon may be shut off from the peritoneal cavity by suturing the bowel to the parietes or very rarely by using a flap from the incised posterior parietal peritoneum to cover the sutured hole in the colon; good drainage must be provided.

By wide mobilization, extensively tattered segments can be exteriorized and removed. Gross tearing of the colon carries a poor prognosis owing to widespread soiling and hæmorrhage. In two cases in this series the colon (1 ascending and 1 descending) was torn so extensively that colectomy had to be performed: both cases died. When a large segment of the colon has been sacrificed, the two colonic stomata should be brought out independently at the most convenient sites. Primary anastomosis is unsound, but, after removal of a hopelessly damaged right colon, a lateral ileo-transverse colostomy, leaving the proximal end of the transverse colon open as a terminal colostomy, may be justifiable.

The retroperitoneal tissues, especially when bruised and hæmorrhagic, are very liable to spread of infection; dissection in this plane, therefore, must be the minimum required for mobilization of the damaged colon. Sulphathiazole powder or sulphadiazine suspension should be used and a separate stab drain is best inserted.

A colostomy can always be closed, but a long slack loop with its limbs tacked together will make closure easier and safer. For this reason, the perforation should be placed at the apex of the loop, when possible. A lesion of the right or left portion of the bowel is extraperitonealized through a separate laterally placed oblique incision, since the loop will then lie more snugly. The thicker the abdominal musculature around a colostomy, the better; the transverse colon should be brought out through the rectus muscle and not through the midline. The same desiderata hold good for a proximal colostomy, which should always be opened at once.

Post-operative Care.—Constant, intelligent, and thorough post-operative care is only possible if evacuation is postponed until there is not only cardiovascular stability, but complete restoration of normal gastro-intestinal activity with a satisfactory fluid and salt balance. The average time the patients were retained before evacuation in a series of 95 patients was 13·5 days.

The main essentials in post-operative care are control of fluid and salt balance by continuous intravenous therapy, decompression of the gastro-intestinal tract by suction through a duodenal tube, morphia for rest of mind and intestine until the joyful tinklings of awakening alimentary activity are heard, and also chemotherapy.

Complications following Operation.—

Extraperitonealization (74 cases).—In this series of wounds of the colon, the damaged area was exteriorized on 71 occasions for penetrating wounds, and 3 times for non-penetrating injury.

Eight cases died of hæmorrhage or shock and multiple injuries within 48 hours of wounding; 1 case died of peritonitis on the sixth day; and 1 case died of pneumonia on the ninth day.

In the remaining 61 penetrating wounds exteriorized there were the following complications directly associated with the abdominal wound and operation:—

Infection:
- 1 pelvic abscess.
- 3 retroperitoneal abscesses (2 deaths after seventh week).
- 1 periproctitis due to wound of lower pelvic colon.
- 1 low-grade peritonitis (death eighteenth day).

Intestinal obstruction:
- 1 due to small loop around colostomy in eighth week.
- 1 due to adhesions (death seventh week).
- 1 case died 10 days after wounding from pyæmia (? infective endocarditis).
- 1 case died on the eleventh day with jaundice and enlarged liver following blood transfusion.

There were 3 cases of multiple wounds of the colon which survived following exteriorization:—
- 1 separate exteriorization of wounds of the transverse and descending colon.
- 1 exteriorization of the whole ascending and proximal transverse colon for wounds of both portions.
- 1 exteriorization of the whole descending colon, splenic angle, and distal transverse colon for wounds of descending and transverse portions.

Suture and Proximal Colostomy or Cæcostomy (17 cases).—Of the 17 cases recorded, 5 were non-penetrating. Of the 12 penetrating injuries, 2 died within 48 hours. Among the 10 remaining, there were the following complications directly associated with the abdominal wound and operation:—
- 2 subphrenic abscesses (1 death in 24 hours).
- 2 infection of the abdominal wall and fæcal fistula (1 death in 3 weeks).

Suture only With or Without Drainage (23 cases).—Of the 23 cases recorded, 5 were non-penetrating. Of the 18 penetrating wounds, 3 died within 48 hours. Amongst the remaining 15 penetrating wounds, there were the following complications directly associated with the abdominal injury and operation:—
- 2 intra-abdominal abscesses—1 subphrenic (death on seventeenth day).
- 2 infections of abdominal wall—1 case with recurrent burst abdomen.

(It is of some interest that in one case with a non-penetrating colon wound, the left common iliac artery had been ligatured; the whole limb survived. The surgeon had injected the lumbar chain with novocain whilst the abdomen was open. The writer saw this case at the Base some months afterwards with evidence of vascular insufficiency—weakness, wasting, and claudication; a left lumbar ganglionectomy was performed.)

Colectomy (2 cases).—Colectomy was performed twice for extensive tearing of the ascending and descending colon. Both cases died.

Drainage only (5 cases).—
1 for peritonitis following a G.S.W. which had produced gas gangrene of the cæcum. The time-lag was 3 days (death).
2 for perforation (?) of the cæcum.
1 for perforation of the ascending colon. Developed a subphrenic abscess.
1 for extraperitoneal perforation of the ascending colon after a time-lag of 10 days—associated pyopneumothorax.

In reviewing these complications, the incidence of local infection following the various procedures is as follows :—

In 61 penetrating wounds treated by exteriorization, there were 6 instances of infection, mainly localized and intra-abdominal (3 deaths).

In 10 penetrating wounds treated by suture and proximal colostomy, there were 2 intra-abdominal abscesses and 2 infected abdominal wounds (2 deaths).

In 15 penetrating wounds treated by suture, with or without drainage, there were 2 instances of intra-abdominal abscess (1 death) and 2 instances of infection of abdominal wound.

In 5 penetrating wounds treated by drainage only there was 1 subphrenic abscess.

The risk of intra-abdominal infection appears to be increased when exteriorization is not performed, though intestinal obstruction does occur in association with a colostomy on rare occasions.

Diaphragmatic Hernia.—Diaphragmatic hernia with acute obstruction of the colon was reported in 2 cases, five months and three years respectively after wounds of the lower chest. In both these cases there had been an uneventful recovery, but later recurrent attacks of undiagnosed upper abdominal pain had occurred before complete obstruction supervened; both died.

It is wise during convalescence to investigate the integrity of the diaphragm in patients who have suffered wounds of the lower chest or possible abdomino-thoracic injury, especially when unexplained recurrent abdominal symptoms are present.

Closure of Colostomy.—The advisability of closing a colostomy depended directly upon the military situation, the available shipping, and, to some degree, upon the length of the sea evacuation route.

A total of 28 colostomies was closed, 17 of these at the Middle East Rectal Centre. In 14 cases sulphasuxidine 20 g. in 24 hours was used pre-operatively and post-operatively, and, when in short supply, sulphaguanidine 3½ g., and sulphathiazole 1 g., were given four-hourly for four days before operation. The majority were closed by means of an enterotome, followed by extraperitoneal mobilization and suture. There were no complications, and very little wound infection, though a few wounds discharged a small amount of fæces for a day or two; all healed well within 10 days.

Latterly the attention of forward surgeons to primary operative details, when justifiable, made closure easier. Some difficulty was experienced when the artificial anus was tucked up under the costal margin or was near the iliac crest, or when there was persistent wound infection with a deficiency in the abdominal parietes or associated osteomyelitis of the ilium or ribs.

One officer, repatriated from Italy, reported that he had suffered a deal of discomfort from attempts by Italian surgeons to "reduce" his colostomy in spite of a good spur ! !

A number of soldiers returned to duty in the Middle East after closure of colostomy.

Sulphonamides.—There can be no doubt of the value of these drugs in the control of infection. It has been the practice of the forward surgeon to place 10 g. sulphadiazine suspension into the peritoneal cavity after operation.

The sulphadiazine suspension is prepared by making a 20 per cent suspension of sulphadiazine in a filtered 3 per cent solution of best gelatine in normal saline (i.e., 10 g. of sulphadiazine in 50 c.c.). It is sterilized for 20 minutes at 20 lb. pressure.

The suspension is injected down a catheter placed into the pelvis, the catheter being then sealed with a spigot.

Absorption takes from four to five days and post-mortem examinations have shown that there is very little local peritoneal reaction to this substance.

Unfortunately, many of the notes available have not given full details of sulpha therapy. Only 84 of the cases analysed had records of sulpha drugs—63 local and 21 general—so that it is difficult to give any convincing evidence of their value when used locally in injuries of the large bowel and rectum.

It is seen, however, that in 17 cases surviving penetration of the colon, after a time-lag of 12 hours, 10 had sulpha therapy (8 local sulphadiazine, 2 general sulphonamides): probably a higher proportion had local sulphadiazine, though no mention was made in the notes.

Further, in the 8 cases surviving after a time-lag of 24 hours, 6 had sulpha drugs (5 local sulphadiazine, 1 general sulphonamide). Of the remaining 2 cases in which no record of chemotherapy was made, 1 was an extraperitoneal hæmatoma of the ascending colon which did not form a fæcal fistula for several days following simple drainage.

Though there is no direct evidence of the value of intraperitoneal sulphadiazine, it is probable, in cases with a long time-lag, that it played a part in recovery.

SUMMARY

1. A total of 128 wounds of the colon, mainly in survivors, are analysed.

a. The right, transverse, and left colon appear to have been injured with equal frequency.

b. The cæcum and transverse colon were most commonly injured by missiles entering from the front, whereas wounds of the fixed

portions of the colon—ascending and descending colon, and flexures—were usually damaged from the back. The sigmoid colon was injured from below, the front, and from the back.

c. Entry wounds in the lower quadrants of the abdomen involved the colon most frequently.

2. The relationship between time-lag, site of colon damaged, and recovery is discussed. Though the number of survivors was approximately equal for each segment of the colon injured, there was a marked fall in the number of survivors with wounds of the transverse colon operated upon after the first 6-hour period.

3. Although, in penetrating wounds of the abdomen, the organ most commonly injured is the small intestine, the incidence of wounds of the colon is also comparatively high: the relative frequency of wounds of the small intestine to colon being 7 : 5.

With wounds of the colon, associated intra-abdominal injury occurred in 42 per cent and was most frequent with injury of the transverse colon (63 per cent).

4. In uncomplicated wounds of the abdomen, shock is synonymous with internal hæmorrhage.

5. Operative approach and treatment of the injured bowel are discussed.

6. Complications following various operative procedures are shown. Closure of colostomy is briefly considered.

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PLASTICITY OF BONE*

BY PROFESSOR JAMES F. BRAILSFORD

IN that part of his lecture on "Diseases of Bones and Joints", which deals with rickets, John Hunter stated, "The bones are often bent by mechanical pressure of the body and by muscular actions, into very peculiar forms and often impede the actions of other parts. The joint forms a more acute angle than it would in health, and then the muscles get a greater power of action. It is most frequent in the back-bone, pelvis, and thigh bones, in the angle made by the cervix femoris, knee, tibia, etc. In these it is most frequent, from the two powers acting (viz., weight and muscular action). The ribs are made straight, or rather bent inwards by the pressure of the atmosphere, so that the child can hardly breathe: a bad formation of the skull will affect the senses: crooked spine will affect the health from an alteration in many of the viscera. In the female pelvis many dreadful consequences occur in delivery. When a bone has admitted of a bias, a disposition takes place on the side unnaturally pressed, for a deposition of bone, so that we find a bony substance formed there which acts as a prop . . . in the thigh the curve is generally forwards towards the upper part, as was seen in an os femoris in which Nature had deposited a quantity of earth on the concave part". In a later part of the lecture which deals with dead bone, he states, "The first effect of the stimulus is on the surface of the living bone, which becomes inflamed: whether new vessels are formed, or the old ones become larger, is undetermined; but by injecting the surface of the part, it appears evidently much more vascular than the other parts. This produces another process. First, absorption of the earthy matter, and all the surface between the living and dead parts of the bone become soft as if steeped in acid, while the dead part remains as hard as ever". In these two brief extracts you have the gist of my paper. My contribution is largely but a confirmation, with the aid of an accessory which Hunter had not, namely X rays, of the observations which he made during clinical investigations. My excuse is that it permits me to emphasize one small part of his teachings which would appear to have been neglected in the employment and dependence on modern ancillary devices, and the neglect of those clinical investigations from which Hunter learnt so much. Used in their proper place, as one can imagine Hunter would have used then, these ancillary services can often help to confirm or dispute theories suggested by clinical investigation.

Features of Normal Bone.—The histologist, Reichert, promulgated the concept that bone, cartilage, tendon, fibrous, elastic, and adipose tissue, derived from the mesenchyme, are all adaptations of or developments from the primitive connective tissue. Subsequently Leriche and Policard brought forward evidence in support of their suggestion that these mature tissues may be induced under certain influences to revert to their primitive state and then undergo ossification. They consider it is only in this way that cartilage or membrane is substituted or transformed into bone. The embryonic connective tissue consists of cells in a collagenous matrix, composed of a fine fibrillar network infiltrated with a very viscid, gelatinous base. The impregnation of this matrix with phospho-calcific mineral matter permits of the setting of the matrix and the formation of bone. The part which the bone-cell takes in these activities has been a matter for considerable discussion. While

* Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on April 26, 1944.

by some it is thought, either by its secretion or by its degeneration, to be the origin of the pre-osseous substance, by others it is assumed that the latter results from physiochemical changes in certain pre-existing connective-tissue elements while the cell exhibits little but osteolytic characters. The normal shape of the skeletal elements is laid down in the primitive mosaic and it has been shown experimentally that, even if these are separated and then cultivated, they develop their general characteristic features. Though the arrangement of the cancellous trabeculæ and the prominence of muscular attachments are modified by function, the general features of the perfect model are preserved by the balance of muscular tension and the strength, hardness, rigidity, and resilience of the bone. The features are changed if the balance is upset, when the relative stresses and strains are diminished or increased relative to the elasticity and strength of the bones. As a result the affected elements bend and certain characteristic deformities are produced unless steps are taken to neutralize the abnormal influence. Normal bone possesses a balance of strength, hardness, elasticity, and rigidity which is determined by the solidity and form of its structure, the condition of the fibrillar base, the comparative amount, cohesion, nature, and condition of its matrix, and the calco-phosphate mineral elements which it holds together.

Prior to the deposition of mineral matter the pre-osseous tissue is plastic and can be compressed and deformed by stresses and strains—the lasting effect of which will be dependent upon the time for which they are continued, the changes wrought in neighbouring tissues, and their shape and position at the time setting of the matrix takes place. With the deposition of crystalline salts of calcium, such as we see in calcification, no material contribution is made to the rigidity or strength of the bone. We see this (Brailsford, 1943) in the skeleton in fœtal osteogenesis imperfecta where all the bones show a granular appearance and multiple foldings indicating a lack of cohesion of the mineral elements — a friability rather than a plasticity. This feature is also seen in one stage of avascular necrosis of bone where the dead fragment collapses or even crumbles within its cartilaginous or membranous envelope. On the other hand, with the incorporation of colloidal calco-phosphate mineral matter, the pre-osseous matrix appears to undergo a rapid setting which may be likened to that of reinforced cement and the bone acquires a rigidity, resilience, and a relative fixation of form which is unaltered by normal function. Normally ossification commencing in an ossific nucleus, spreads uniformly and gradually from the regular centre to the periphery of the cartilaginous mass. It is not deformed by the normal stresses and strains of function of the growing child.

Effect of Irregular Ossification.—If, however, the ossification in any site is irregular and multiple irregular ossific centres develop throughout the cartilaginous structure, this structure will not be capable of withstanding normal function, and, because with the irregular deposition of mineral matter it has lost its elasticity and taken on the characters of a plastic substance, it can be compressed and deformed. Examples of this (Brailsford, 1944) can be seen in hypothyroidism. In the fœtus and young infant with this deficiency the ossification of the vertebral bodies is irregular and the keystone of the general spinal curvature, which is in the dorso-lumbar area, may collapse, resulting in stunted growth of the affected body and the development of a kyphos; while in the older child the femoral capital epiphysis, which exhibits a similar defect in ossification, is splayed out over the upper end of the diaphysis so as to resemble the appearance of healed or healing Legg-Perthes' disease or deformed into the beak shape which is most commonly seen in cretins. In the condition to which I gave the name chondro-osteodystrophy, we also have multiple ossific nuclei; consequently in this disease primary and secondary centres of ossification will be deformed according to the pressures or muscular strain to which they are subjected during the irregular incomplete phase of ossification. In a later portion of the paper more attention will be paid to similar deformities of the skeleton due to localized plasticity induced by trauma or disease.

INFLUENCE OF PARALYSIS ON THE SKELETON

With the cessation of function occasioned by paralysis there occurs a gradual decalcification of the bones of the affected part; eventually all the cancellous tissues within may be absorbed and the compact cortex considerably reduced in thickness; yet with the patient immobilized little change occurs in the outward form or position of the bones. Compared with the opposite normal limb a concentric atrophy of the shafts may later be seen which is emphasized by the more normal sized extremities. The spine under those circumstances is not subjected to defective balance and the normal curvatures are more or less preserved. With the patient up and about with one paralysed lower extremity, the bones of the latter appear to be affected by a gravitational pull. The angle of the femoral neck with the shaft is straightened out and dislocation may occur (Brailsford, 1944). The os calcis and other bones of the foot also show the effect of gravity. Scoliosis of the spine may occur, but the most marked cases of moulding associated with abnormal curvature have developed in children following one of the exanthemata of childhood. Soon after recovery from, say, an attack of measles, a slight curvature of the spine is noticed. The radiographs at this time may show curvature unassociated with any secondary moulding. With the alteration in balance, possibly associated with an undue weakness of the

spinal musculature, the curvature progressively increases in spite of fixation in the spinal jacket. The vertebral column with its processes and ribs undergoes very considerable moulding and the most extreme form of deformity may ultimately result.

The following is an example of this type of case :—

M. B., female aged 6, in 1927 was found to have a left dorsal scoliosis, the lower curve of which appeared to be corrected by suspension (*Fig.* 273). She was put into a plaster case and in 1929 her report

FIG. 273.—Scoliosis in a girl aged 6 years (Sept. 12, 1927) which appeared to be corrected by suspension.

card stated that " it appears to be holding her well ". On Jan. 30, 1929, the general alinement of the body was noted as good. On Aug. 14 plaster was removed and a note stated that the deformity was not increasing. Block leather support with double crutches were provided, and on Oct. 8, 1930, the report states: " Is standing better now "; on Nov. 9, 1932, the report states deformity stationary. Similar reports continue to record the clinical findings, but examination of the series of radiographs taken regularly since the condition was first recognized show a progressive development of the deformity and now the curvature is so extreme that the upper border of the 11th dorsal vertebra is on the same plane as the lower border of the 3rd (*Fig.* 274).

The progressive development of this extreme form of curvature indicated that neither the plaster case nor the block leather supports were able to check the deformity. Immobilization in bed during the early stages, perhaps for two years, with exercises, may correct the balance and so prevent the progressive development of the curvature.

PLASTICITY DUE TO GENERALIZED CHANGES IN THE BONE

Those cases of osteogenesis imperfecta which survive birth and live to adolescence or adult life show in the early months and years a skeleton rather lacking in density but otherwise of normal shape and size. Depending on the gravity of the disorder, we see the development of changes (Brailsford, 1943). More pronounced than in the paralysis, we see a concentric atrophy of the shafts of long bones, while the diaphysial extremities appear to be markedly expanded by

FIG. 274.—Same patient as *Fig.* 273, 17 years later. Note the very considerable advance in the deformity and moulding of the ribs and spine.

their normal development. At this stage the skeleton exhibits a marked plasticity and develops deformities, the extent of which in any part are dependent upon the gravitational and muscular pulls. The head takes on a tam-o-shanter form with its vertex and sides tending to gravitate over the base—the latter tending to fold over the upper part of the spine. The vertebral bodies are compressed to half or one-third of their depth—though the discs preserve or even appear to exceed their vertical dimensions.

The vertebral epiphyses appear to be activated to earlier ossification, so that ossific nuclei may be readily demonstrated in them as early as the sixth year. The ribs show a marked gravitational bending: from their necks they become sharply directed downward. The bones of the upper extremity may almost or entirely escape in the lesser degrees of the dystrophy, but in the severe

forms the shafts of the long bones are slender and exhibit bending due to the superincumbent weight on the plastic bone during crawling. But it is in the lower extremity that we see the major deformities of the plastic bone. The pelvis may be so plastic that it is compressed and distorted

FIG. 275.—Marked plasticity of bones in infantile rickets.

by the multifarious gravitational and muscular pulls until it assumes a wind-swept appearance. The femora exhibit large extremities composed of wide-mesh cancellous tissue with but a thin periphery, while the shafts gradually become slender rods of compact bone (Brailsford, 1944). In their earlier stages of development such shafts are relatively brittle, but later, bending, in spite of muscular atrophy and an increased proportion of compact relative to cancellous tissue, indicates a degree of plasticity of the bones. So deformed may these plastic bones of the lower extremity become that the limbs are functionless, and their encumbrance has caused the surgeon to consider amputation advisable. In the less severe degrees of the dystrophy condensation of bone may occur at the apex of the curvature—Nature's attempt to limit it. In the active phase of infantile rickets the bone is lacking in mineral matter and all ossification is irregular, so that a considerable degree of plasticity of the bone exists which permits of bending and compression deformity of all parts of the skeleton by stresses and strains which would not affect normal bone (*Figs.* 275, 276). With healing of the condition Nature attempts to correct the deformity or prevent its greater development by buttressing the concave aspects of the curvature with stout compact bone, as recorded by Hunter. The deformities which occur in infantile rickets are copied in the adult akeleton with the development of osteomalacia (Brailsford, 1944), but in this adult condition the deformities become more profound owing to the increased stresses and strains to which the adult skeleton is subjected.

In the rickets associated with renal fibrosis, two types of change are seen (Brailsford, 1944). In *Type A* the bones appear to preserve their strength except at the metaphyses—these may disintegrate under pressure and permit of serious displacement. In *Type B* there appears to be an added factor, probably associated with hyperparathyroidism, since in all such cases one has found at post-mortem hypertrophy of all the parathyroids ; this leads to softening of all bones ; they become so plastic that extreme degrees of gravitation deformities may develop, and though the metaphysial areas exhibit an even greater destructive change than in Type A, these sites are not subjected to the displacements we see in Type A.

In hyperparathyroidism associated with a parathyroid tumour a profound decalcification of the skeleton ultimately occurs, leading to marked pressure deformities, but, except in young persons, as the condition of the patient before marked plasticity occurs has been reduced

FIG. 276.—Pressure deformities of the plastic bone of the pelvis and femora in infantile rickets. The active phase has passed.

to almost complete invalidism following multiple fractures and general weakness, the gravitational deformities seen in osteomalacia are not usually present. The type of decalcification in the earlier phases is distinctive (Brailsford, 1944) and its recognition of the utmost importance for early diagnosis before deformity or fracture occurs. Though in the latter stages it is associated

with destruction of large areas of cancellous tissue and the production of multiple cyst-like structures, which has earned for it the title 'osteitis fibrosa cystica', in the earlier stages these cyst-like areas may not be found—there may be but a stippled osteoporosis of the bones with perhaps small areas of ill-defined cancellous absorption.

PLASTICITY DUE TO CHANGES WHICH MAY BE LOCALIZED TO ONE AREA OF THE SKELETON

The most notable of the conditions in which plasticity is exhibited originally in one bone, while all other parts of the skeleton may be normal, is Paget's disease. Unfortunately, the novice who has not mastered Paget's description of the disease, which he called 'osteitis deformans', is apt to interpret the radiographic appearances of hyperparathyroidism as Paget's disease. This has resulted in very serious delay in diagnosis of a condition which, diagnosed early, can be treated with success, but which, after the crippling effects of fractures and invalidism, yields a relatively poor result to treatment. Hyperparathyroidism is essentially a generalized condition from the first, whereas Paget's disease begins as a localized condition. Paget's description of the condition should be reproduced in all text-books (Brailsford, 1944), for though he was, perhaps fortunately, without the aid of radiography and more recent laboratory investigations which appear to detract attention from the patient, his clinical observations were so accurate and exhaustive that little addition has been made to our knowledge by the more recent developments.

Radiographic study of the skeleton in Paget's disease reveals that the condition may commence in one bone or in several widely separated bones. It may commence as a subperiosteal change which on the radiograph in the early stage is represented as a lanceolate cyst-like absorption of an area of the cortex of a bone which is elsewhere normal—the line of demarcation between the Paget lesion and the normal bone being fairly sharply defined; progress of the lesion is indicated by stripping up of the periosteum by a V-shaped thrust into the normal tissue.

When the whole periphery of the bony segment has become involved the normal compact cortex within is gradually absorbed and the bone develops an increased plasticity which permits of bending under ordinary physiological strains. Gradually the whole bone is changed. The rate of progress may be so slow that it may be 10–15 years before the whole bone is involved. This is important from a medico-legal aspect when the patient alleges that the limb was normal until a recent injury. The disease may remain localized to the one bone for years, or it may then spread across the joint and involve adjacent bone without producing any appreciable alteration in the articular surfaces. In one case one rib was involved and the disease involved its vertebra and then its fellow on the opposite side—the other bones remaining normal. In the same patient the disease may commence in another bone as a localized endosteal change—here the cancellous spaces appear to be filled up with a mineral deposit giving the bone greater and uniform density. In the smaller bones and the flat bones all distinction between compact and cancellous tissue may be lost in the uniform density, but in the larger tubular bones like the femur a thickened cortex may give the bone the appearance of marked hypertrophy. Such changes may occur without attracting the serious attention of the patient, i.e., serious enough to seek radiographic examination.

I have shown (Brailsford, 1944) that the radiographic appearances of Paget's disease may exhibit three distinct types of bone changes. To these I have given the names (1) Osteoporotic, (2) Osteolithic, (3) Lithocystic. The first type shows general expansion of the bone, osteoporosis, coarse trabeculation, and bending under normal physiological strains. In the acute phase the bone is plastic and marked bending may be unassociated with any breach of the surface, but complete fracture may occur during simple unguarded movements. At a later stage the long bones exhibit the reaction to strain which we see in a banana when its curvature is increased by pressure. The concave aspect is condensed while the convex aspect shows one or multiple fissures running towards the centre from the periphery, i.e. incomplete fractures.

The osteolithic bone is much denser than normal and of stouter build, and though it gradually bends under continuous physiological strain it exhibits an abnormal fragility and may snap like a stick of chalk with trauma insufficient to damage normal bone; during the following 2–3 weeks it may develop acute halisteresis. This form is simulated by carcinomatous infiltration of the bone from primary carcinoma of the prostate, stomach, or oesophagus, though in these, because of the more rapid reaction, condensation of the bone is not associated with the degree of plasticity and consequent expansion and deformity seen in Paget's disease.

In the lithocystic form, which in its early stage may show marked decalcification, the affected bones eventually exhibit massive development with thickening of the dense cortex, within which cyst-like areas of various sizes without cancellous trabeculation may be recognized. In spite of the massive build of these bones considerable bending may occur and transverse fracture may occur as with the osteolithic type. The disease not only involves the normal bones, but ossified ligaments, massive repair bone at the site of old injury or old inflammatory foci, and ectopic bone in the soft tissues, develop the Paget characters of adjacent bone. In many cases biopsy has been performed to establish the diagnosis, but nothing has been added in

all these years to the description of the histological appearance included by Paget. Far more can be learnt of the condition by careful periodical X-ray examinations of the skeleton. The more attention we pay to these serial radiographic studies the more readily we can anticipate the histological appearances. I feel that this is the method which Hunter would have used. Even without the urge of use of the multifarious ancillary services which we have to-day, Hunter's experience caused him to state, " It is astonishing to see what little curiosity people have to observe the operations of Nature, and how very curious they are about the operations of Art."

There is a condition called by Elmslie ' fibrosis of bone ', or more recently by Lichtenstein and Jaffe ' polyostotic fibrous dysplasia ', in which

FIG. 277.—Patient with generalized polyostotic fibrous dysplasia.

the skeleton of the individual gradually develops, locally or throughout its whole structure, a remarkable plasticity permitting of great deformity (*Figs.* 277–279). The localized lesions as they occur in the skull or long bones exhibit radiographic appearances which may be mistaken for those of simple cysts—the lesions are represented by clearly defined expanded segments of bone which are devoid of cancellous trabeculæ but yet they are denser than the simple cysts. Such conditions have also been mistaken for those of hyperparathyroidism, and operative measures for the removal of parathyroid tumour have been undertaken in many cases. The distinguishing feature is that all other parts of the skeleton present the appearance of normal bone, even that adjacent to the so-called cysts shows normal cancellous and compact tissue—there is no general decalcification. In those

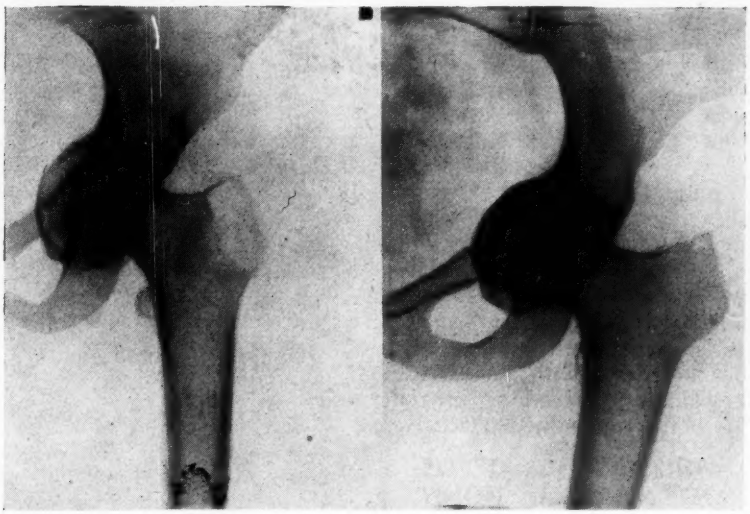
FIG. 278.—Radiograph of the legs of patient shown in *Fig.* 277. Note marked bending of the bones.

cases which are more generalized the defective segments show whorls of chondrous tissue, with or without calcification of their peripheries

FIG. 279.—Radiograph showing polyostotic fibrous dysplasia in the one limb only.

(*Figs.* 278, 279). The localized areas are expanded, in the skull producing an appearance which may be mistaken for leontasia ossea, and in the long bones permitting of bending, particularly when the lesions are developed in the lower extremity.

Localized inflammatory lesions in, or in the neighbourhood of, bones, lead to decalcification and plasticity which permits of marked deformity, unless measures are adopted to prevent it. The development of coxa vara following inflammatory lesions in the femoral neck is an instance.



A B

FIG. 280.—A, Protrusio acetabuli a few months after subacute arthritis of the hip-joint. B, Same hip-joint 10 years after. Consolidation of the plastic bone and no further extension in the interval.

Following trauma to the hip-joint, or as the result of inflammatory lesions in or around the hip-joint, the walls of the acetabulum become plastic for a time and if subjected to pressure during this phase will be pushed by the femoral head into the pelvis—thus is produced the lesion referred to as protrusio acetabuli. Consolidation of the protruded bone is brought about by the deposition of much mineral matter, and unless the inflammatory reaction persists the deformity is stabilized. Observations of such cases over 10 years have shown little change (*Fig.* 280). This suggests that the deformity might be prevented by immobilizing the patient during the active inflammatory phase.

FIG. 281.—Bowing of the femoral shaft in spite of plate. Screws pulled out of the plastic bone during bending.

As I showed in a previous paper (Brailsford, 1936), plasticity of bone may develop after union of fragments which had been fixed by metallic plates or pins, and the fixing screws or other fittings be torn from the plastic bone (*Fig.* 281). This reaction in the bone has been attributed to electrochemical changes between the bone and the metal. Though attempts have been made to check it by using metals considered free from this action, my radiographic experience suggests that removal of the metallic support as soon as the radiograph shows bony union between the fragments, followed by a short period of immobility, would give the better results.

CHARACTERISTIC CHANGES IN VARIOUS SKELETAL ELEMENTS EXHIBITING ABNORMAL PLASTICITY

It is impossible in the brief time at my disposal to do more than indicate the characteristic deformities which arise from abnormal plasticity. Their significance (Brailsford, 1944) cannot be discussed.

The Skull.—The most important of the pressure deformities is that referred to as the basilar impression—it is most commonly associated with Paget's disease and has been said to be associated with the carrying of heavy weights on the head.

The Spine.—Localized plasticity of the vertebral column as the result of injury or disease comes to be associated with compression myelitis. Scoliosis and kyphosis and kyphoscoliosis are all eventually associated with moulding and condensation of compressed bone.

Biconcave compression of the vertebral bodies, until they simulate fish vertebræ, with relative expansion of the discs, is seen more commonly in osteogenesis imperfecta, the various types of rickets, osteomalacia, and hyperparathyroidism.

The Pelvis.—Besides the complicated distortions of the plastic pelvis such as we see in osteogenesis imperfecta, Paget's disease, and osteomalacia, localized sites of osteochondritis occur in the neural arch of the 5th lumbar. This may be associated with the progressive deformity of spondylolithesis, as was seen in the specimen which used to be in the Hunterian collection. Protrusio acetabuli, as we have seen, is due to gravitational strain on the head of the femur, against an acetabulum the pelvic wall of which has been softened as the result of post-traumatic inflammatory changes and subacute arthritis—particularly post-menopausal arthritis. It also occurs in Paget's disease of the pelvis.

Limb Bones.—The chief deformities of the limb bones are those associated with an increase in the curvature producing alterations in the normal angles of the bones and joint surfaces.

CONDITIONS ASSOCIATED WITH REDUCTION OF NORMAL BONE ELASTICITY

In Albers-Schönberg's disease, certain forms of Paget's disease, and secondary carcinomata of the skeleton, though the affected bones are abnormally dense from their increased mineral content, they appear to have lost the characters associated with normal fibrous bone and transverse fractures occur as the result of trauma insufficient to fracture normal bone. In tabes dorsalis, though the bones appear to be of normal radiographic appearance, they often exhibit an unusual fragility and comminuted fractures occur; the lack of muscular protection is probably a feature in these cases (Brailsford, 1941).

LOCALIZED PLASTICITY OF BONE DUE TO TRAUMA

When a bone is subjected to persistent abnormal strain, reactive inflammatory changes are incited, localized softening of the bone occurs, and pressure deforms the plastic bone. This phenomenon is seen more commonly perhaps in the spine than in any other region. In any occupation, particularly in young individuals with incomplete ossification, which necessitates continuous bending, compression is exerted on the anterior aspects of the dorsal spine. With the healthy individual physiological periodical reshuffling adjusts the tissues and no harm is produced, but in the debilitated and fatigued person such reshuffling is diminished or even abolished. Consequently the pressure becomes continuous and reactive inflammatory changes are induced in the tissues under compression. Some localized decalcification with plasticity results. Later re-ossification fixes the deformity. Continued pressure leads to condensation of the opposing bony surfaces and ossification of the adjacent ligaments—Nature's method of checking the growth of the deformity. Any lesion, whether it be developmental, traumatic, inflammatory, or neoplastic in origin, which alters the

FIG. 282.—Scoliosis with marked lipping of the vertebral bodies in the concave aspects of the curvature.

normal alinement or balance of the skeleton, will cause abnormal strains to be placed on some structure; sooner or later this will show the reactive changes detailed above (*Fig.* 282). With the young person no disability or reactive changes on the radiograph may be detected because of possible physiological compensations, but as the strains of adult life begin to mount, symptoms develop and serial radiographs show a progressive change. A good instance of this is the young patient who develops a pneumococcal arthritis of one hip-joint which goes on to bony ankylosis. After a year or so he has accustomed himself to the normal fixation and certain compensatory activities have developed, but in the late 20's or early 30's the opposite hip-joint, which was normal, will show abnormal moulding of the roof of the acetabulum and later some condensation of the subarticular bone. Within a few years atrophy of the superior articular cartilage will be apparent, followed by the progressive development of osteo-arthritis—a very serious surgical problem in so young a person.

PLASTICITY AND OSTEOCHONDRITIS

Most of the secondary and some of the primary ossific centres develop the changes which have been included under the general heading osteochondritis. In the initial stages of the condition, when associated with necrosis of well-formed ossicles, the young bone develops an increased density and friability. As a result the affected bone is crushed or fragmented, but

its articular cartilage is apparently spared (*Fig.* 283). Later the interstices between the fragments appear to be infiltrated with living tissue which brings about gradual absorption of all the mineral matter from the fragments (*Fig.* 284). During the whole of this phase the affected bone is plastic and can be deformed by pressure from adjacent bones. But the necrotic fragment is not the only bone which is affected. Reactive inflammatory changes occur in the adjacent living bone. It is decalcified, apparently at the expense

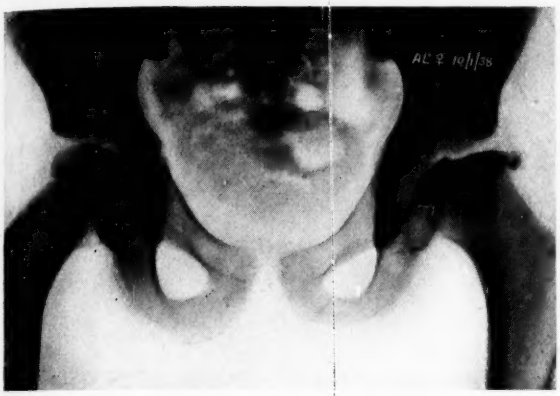


FIG. 283.—Active plastic phase of Legg-Perthes' disease of the femoral capital epiphysis.

of the dead fragment, and it also becomes so plastic that it can be deformed by pressure. As the dead fragment is progressively decalcified and reconstituted, the adjacent bone recovers its calcium and normal characters, but any deformity produced during the plastic stage is fixed. It will be obvious therefore that if we are to avoid deformity, and the subsequent series of changes leading to osteo-arthritis, we must protect the plastic bone from any pressure. As I have shown, the sequence of changes which such bones undergo have a time table which permits us to arrange for the necessary immobilization. For the condition known as Legg-Perthes' disease I draw up the following time table from a study of the serial radiographs of a large group of cases.

FIG. 284.—Same patient as in *Fig.* 283, fifteen months later, showing further compression of plastic bone and some absorption of calcium.

It shows that for upwards of 3–4 years the bone exhibits abnormal plasticity. Until the last dense island has been absorbed this plasticity will permit of deformity by pressure. In certain other sites, such as the infant scaphoid and the vertebral body, the time table is speeded up, and within 1–1½ years the affected bone has been sufficiently reconstituted to permit of normal function. The cause for these localized lesions of osteochondrosis is not definitely known. Trauma and embolism have been suggested amongst other things. Undoubtedly trauma can produce avascular necrosis of fragments of bone.

ONSET OF RADIOGRAPHIC APPEARANCES IN LEGG-PERTHES' DISEASE

RADIOGRAPHIC APPEARANCES	AGE OF LESION JUDGED FROM ONSET OF FIRST SYMPTOMS	CONDITION OF THE BONE
1. An increase in the density of the femoral capital epiphysis 2. A relative increase in the joint space 3. Osteoporosis of the diaphysis 4. Compression and impression fractures of epiphysis	2–3 months	Becoming plastic
5. Appearance of fragmentation 6. Compression and flattening of fragments 7. Gradual absorption of the dense fragments 8. Compression and expansion of the proximal end of the diaphysis	3–18 months	Plastic
9. First appearance of regeneration in the epiphysis in which dense fragments are undergoing absorption 10. Absorption of the last dense nucleus, and the appearance of a circumscribed area of osteoporosis in its place 11. Increased deposition of calcium and obliteration of the osteoporotic zone in the diaphysis	1½–4 years	Plastic
12. The epiphysis assumes the radiographic appearance of a normal bone	During or after the 4th	Consolidated

We see it more commonly following fracture of the femoral neck or carpal scaphoid. The most important feature is that these necrotic fragments

A B C

FIG. 285.—A, Fracture of the femoral neck. Disturbance of vascularity of head fragment is indicated by its relative density. B, The same six weeks later. C, The same six months later. Radiographs at three months were regarded by the surgeon as indicating union, and patient encouraged to use limb. Note the coxa vara deformity which has resulted owing to pressure on the plastic bone. The head fragment was reconstituted in four years.

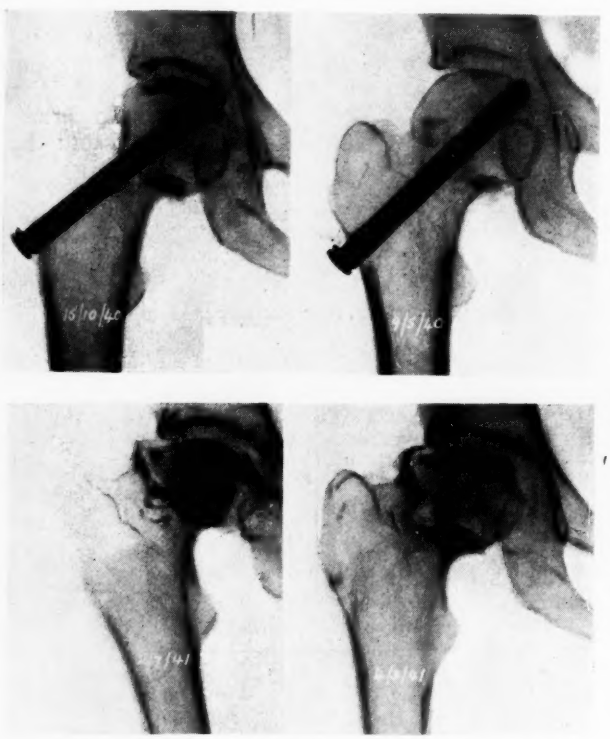


FIG. 286.—Series of radiographs illustrating collapse and moulding of avascular head fragment of femur.

excite inflammatory reactive changes in the adjacent vascular bone leading to its decalcification and abnormal plasticity, until the necrotic fragment has been removed or entirely reconstituted (*Fig.* 285). It has been stated by one writer on fractures that "There is no such thing as osteochondritis; it is avascular necrosis", but the radiographic evidence shows that the most important feature associated with necrotic bone is the accompanying osteochondritic reaction in the adjacent living bone (Brailsford, 1943). Failure to appreciate it has led to very many disasters following pinning of the fractured femoral neck. The fragments were adjusted as near as possible to normal alinement and held in position by a Smith-Petersen pin or other metal nail. After an interval, sometimes but a few days or weeks, or perhaps even as long as three months, the patient was allowed to use the limb (*Fig.* 286). Because the radiograph appeared to the observer to show good alinement and perhaps some union, the patient was encouraged to get the limb into full function, i.e. he was rehabilitated. Following such an injury a certain amount of pain was experienced in using the limb, but if this was mentioned, the surgeon, supported by his reading of the radiograph, explained that this was to be expected for some time. The patient gave progressively increased function to the limb and because the surgeon had indicated that he expected some pain and disability this was ignored, until, after a year or more, these features became so pronounced that further advice was sought. Radiographs now showed that the pin had cut through the plastic bone of the femoral head or neck or even the walls of the acetabulum, as it would

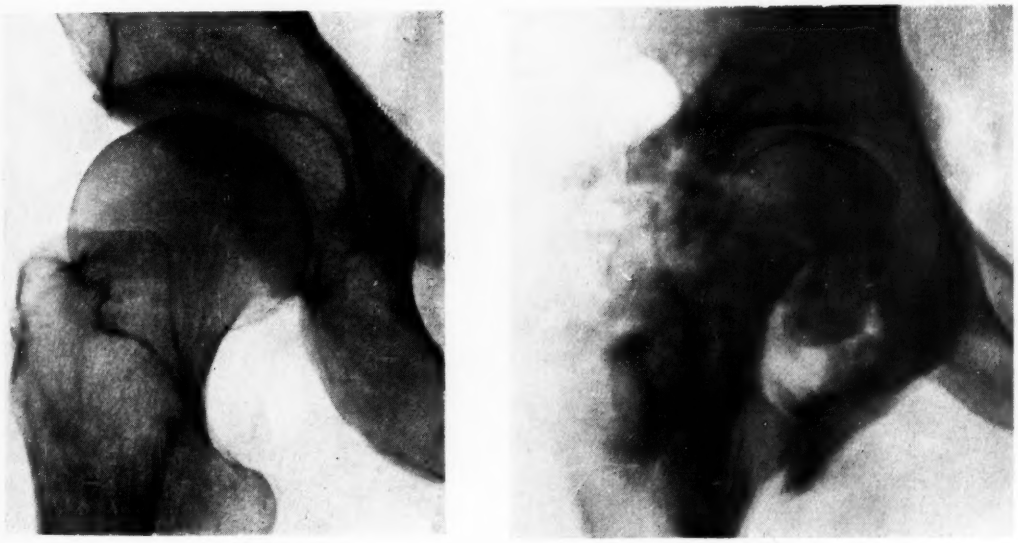
through butter, because of the superincumbent weight of the trunk on the pin (*Fig.* 287). Many disasters of this type have been used to illustrate the changes in avascular necrosis. Similar disastrous sequelæ have followed—rehabilitation of a dislocation of the hip-joint in which radiographs three months after the injury have been erroneously interpretated as normal (*Figs.* 288, 289). The possibility of progressive absorption in tabes should be borne in mind (*Fig.* 290).

A B

FIG. 287.—A, Radiograph showing pinning of neck of femur three months after insertion of pin. Radiograph interpreted as showing union of fragments. Function permitted. B, Same patient. Note the wide excavation of the plastic bone of the femoral neck and displacement of fragments and pin.

In a previous paper I pointed out that unless the patient with an avascular fragment can submit to immobilization until radiographs show reconstitution of the fragment, i.e., upwards of three to four years, the necrotic fragment should be removed as soon as detected. This opinion I had formed from a study of several hundred cases of osteochondritis associated with and without avascular necrosis.

In the preparation of this paper I again studied the lectures of Hunter and I found he had given us the explanation from his careful clinical examinations. He states " When a piece of bone becomes absolutely dead, it is then to the animal machine as any other extraneous body, and adheres only by the attraction of cohesion to the machine. The first business of the machine, therefore, is to get rid of this cohesion



A B

FIG. 288.—A, Reduced dislocation of hip-joint. A little calcium below femoral neck. B, Same two years after, showing disintegration. Avascular head fragment and much ectopic bone.

FIG. 289.—Marked compression deformity of femoral head and neck following surgical manipulation and reduction of congenital dislocation.

and discharge it. For affecting this separation there are several natural and successive operations going on. The first effect of the stimulus is on the surface of the living bone which becomes inflamed; whether new vessels are formed, or the old ones become enlarged is undetermined; but by injecting the surface of the part it appears eventually much more vascular than the other parts. The surrounding parts also inflame, as the periosteum and cellular membrane, and often take on an ossific inflammation. This produces another process; first, absorption of the earthy matter, and all the surface between the living and dead parts of the bone become as soft as if steeped in acid, *while the dead part remains as hard as ever*. To complete the separation, the absorbents continue their office and absorb the living parts also". These findings of Hunter are confirmed by my radiographic investigations.

Had his teaching been fully appreciated, recent faulty interpretations would have been avoided and patients have been spared some of the disasters attendant upon necrotic bone.

In the question of the dead bone remaining "as hard as ever", Hunter was considering the question of the sequestrum in sepsis. In aseptic necrosis it applies only for a short time, as will be apparent from the radiographic evidence of collapse and friability of the dead fragment within a few months of its death. More recently

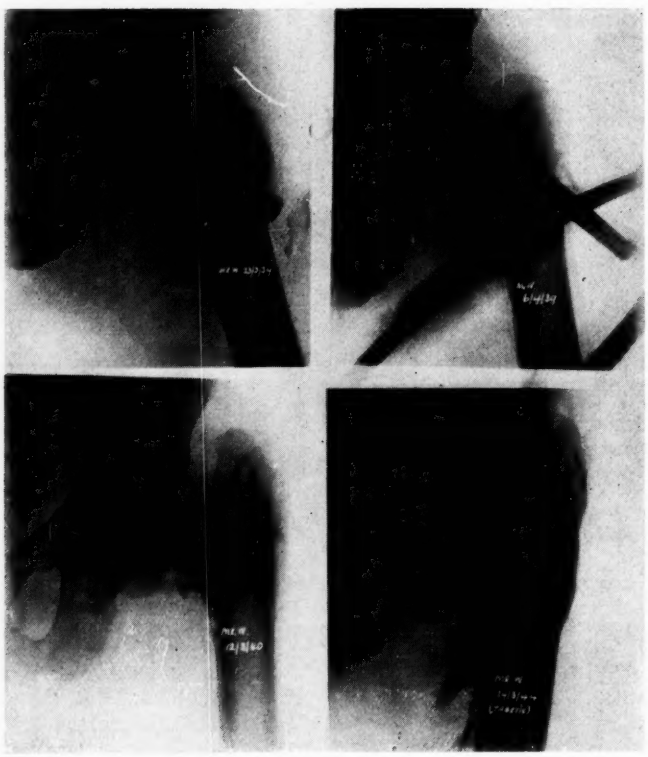
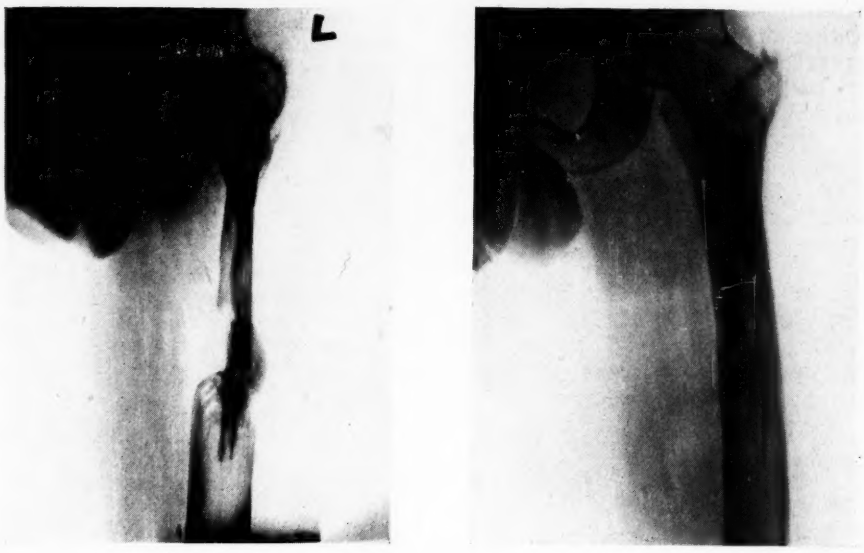


FIG 290.—Progressive absorption and moulding in a case of tabes. Note the development of the Charcot's joint at the fracture site.

Leriche and Policard have stated, " Physiologically dead bone, with dead cells, seems to have the same mechanical value as when it is living. But as long as it is tolerated and as long as it is maintained in the organism, it seems to play the same mechanical part ". This opinion is not borne out by radiographic evidence, for the dead fragment sequestrated by inflammatory changes retains its hardness and sharpness of outline, whereas the fragments tolerated tend to undergo progressive decalcification and softening, and induce reactive changes in the adjacent bone which lead to plasticity as a preliminary step to reconstitution and consolidation (*Fig.* 291, A, B).



A B

FIG. 291.—Radiographs showing reconstitution, consolidation, and growth of bone-graft replacing polycystic lesion. A (May 5, 1938), Shows callus around fracture of lower third of graft (shaft of fibula). Decalcification of femoral shaft. B (June 4, 1944), Shows consolidation and growth of graft, which now equals the calibre of the femoral shaft. (Mr. A. M. Hendry's case.)

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CONGENITAL DEFICIENCY OF THE PERICARDIUM ASSOCIATED WITH A BRONCHOGENIC CYST

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INTRODUCTION AND HISTORIAL SURVEY

IN the year 1788 Matthew Baillie opened the chest of a man of about 40 years and saw a naked heart lying in the left side of the chest. In reporting this case in 1793, Baillie gave the first authentic account of congenital deficiency of the human pericardium, a condition so seldom encountered that even to-day it can be truthfully described in Baillie's original words as " one of the deviations from the ordinary structure of an animal in which nature has been most sparing ".

Some earlier examples of the condition have been reported, the first by Realdus Columbus in 1559 (Curling, 1839), and others by Littré in 1712 and Vieussens in 1715. Vieussens described the findings at autopsy upon a soldier, aged 35, who had two hearts, one of which was devoid of pericardium. Upon the validity of many of these earlier case reports (which can be found summarized in the publications of Otto in 1831 and Curling in 1839) doubt has been cast by anatomists on the grounds that they were examples of diffuse adhesive pericarditis and not of genuine pericardial deficiency. Baly reported another case in 1850 in a young man in whom autopsy

disclosed the heart lying in the left pleural cavity invested by the visceral pleura. Four years later Bristowe (1854) published another case of a male aged 28 in whom the heart and left lung occupied a common serous cavity. Peacock, in the second edition of his *Malformations of the Human Heart* (1866), was familiar with the publications of Baillie, Curling, and Baly cited above, and had himself observed an example in a man aged 75 who had died of aortic valvular disease. The heart lay in the left pleural cavity adherent to the left lung. In contrast to the foregoing examples of complete absence of the pericardium, Powell in 1869 recorded the interesting finding of a partial deficiency. The patient died from spontaneous pneumothorax on the left side, and post-mortem examination disclosed air not only in the pleural space but in the pericardium also, the two serous sacs communicating through a small opening with a smooth edge lying above in the left lung root.

During the next 40 years other cases were placed on record, and Ebstein in 1910 compiled 32 examples up to that time. In 1925 Moore undertook a comprehensive study of the available medical literature and collected 64 recorded cases, obtaining access to the protocols of 42 of them. They had all been discovered accidentally at autopsy.

A careful search of the literature since the publication of Moore's paper has brought to light 15 further examples occurring in man, all of them, with 2 exceptions, being chance findings post mortem. These two exceptions have been provided by Ladd (1936) and Dahl (1937). Ladd operated upon a left-sided diaphragmatic hernia in a 2-year-old girl, using an abdominal approach. Through the hole in the diaphragm he saw that the heart was without a pericardium, but the existence of a rudimentary tag at the base could not be excluded. King (1942) states that he has had a similar experience, but gives no details. Dahl inferred the presence of a communication between the pericardium and left pleural space during artificial pneumothorax treatment for pulmonary tuberculosis in a man aged 20, from a radiograph taken soon after the induction, which revealed a pneumopericardium in addition to a left pneumothorax.

We are prompted to place another example on record not so much on account of the abnormal pericardium, but because it was found together with an unusual developmental anomaly of the lung, an association which may shed some light upon the mechanism of the occurrence of partial deficiency of the pericardium. The case we are about to describe is the first to be directly approached and closely studied during life.

CASE REPORT

HISTORY.—K. M. O'L., female, unmarried, aged 19, attended the Out-patient Department of the London Chest Hospital on Aug. 9, 1940, complaining of pain in the upper part of the left side of the chest for the past six months. It was described as a dull ache, unrelated to respiration. An unusual degree of breathlessness had been experienced during fairly strenuous exertion, but in the ordinary way her breathing had been unremarkable. She had lost 5 lb. in weight in the preceding three months. There were no other symptoms: no cough and no sputum. General health was good; she felt full of energy; and had been able to do her secretarial work without discomfort.

Past medical history had been completely uneventful and the family record was good.

ON EXAMINATION.—Well-built, healthy looking girl. No clubbing of the fingers and no glands palpable. Apex beat in the normal situation. Faint pulsation could be felt in the 2nd left interspace near the sternum. A soft systolic murmur was heard at the apex and a coarser bruit at the pulmonary area. The pulmonary second sound was accentuated and reduplicated. Blood-pressure was 112/72 and equal in the two arms.

Movement of the left upper part of the chest was defective; there was some impaired resonance in the 2nd and 3rd left interspaces close to the sternum; and the breath-sounds on the left side below the clavicle were harsh. No adventitious sounds were heard and there were no physical signs in the chest behind. Other systems were normal.

Radiology: Anteroposterior X-ray picture of the chest (*Fig.* 292) showed a globular shape of heart with a curious thumb-shaped opacity with a smooth outline spreading out from the mediastinum in the neighbourhood of the pulmonary artery. A left lateral view (*Fig.* 293) showed this opacity to be anterior in position.

A provisional diagnosis of a pulmonary cyst or mediastinal tumour was made and the patient was admitted for further investigation on Aug. 30, 1940.

Temperature was normal except for an occasional ascent to 99° F.; pulse-rate ranged from 80–90 per minute; respiration rate was 20.

Fluoroscopy: The mass pulsated, but was distinct from the aorta. It was closely related to the pulmonary artery. There was no enlargement of any of the individual chambers of the heart.

A *kymogram* confirmed the pulsation of the mass.

Electrocardiogram showed normal curves.

Other tests such as blood Wassermann reaction, Friedman test, and blood-count were unhelpful.

When the bombing of London started the patient left hospital before investigations were completed.

She attended the Out-patient Department during the winter of 1940–41, and in November, 1940, she returned to work. There was slight unproductive cough. The pain in the chest sometimes radiated down the inner side of the left arm. Breathlessness was unchanged and she had gained 2 lb. in weight. Physical signs and radiographic appearance were the same.

She continued thus until July, 1941, when the pain increased in severity and penetrated to the back; she experienced "choking sensations", and there was some failure of the general health. There was no alteration, however, in the physical findings, and the X-ray picture showed no increase in the size of the tumour.

As subsequent investigations shed no further light upon the aetiology of the condition, the decision to explore the chest by thoracotomy was reached. A preliminary pneumothorax was induced upon the left side and a radiograph (*Fig.* 294) showed that the lung

had collapsed and that there was air in the pericardium as well as in the pleural space. The full significance of this observation was not appreciated at the time and the operation was undertaken as planned.

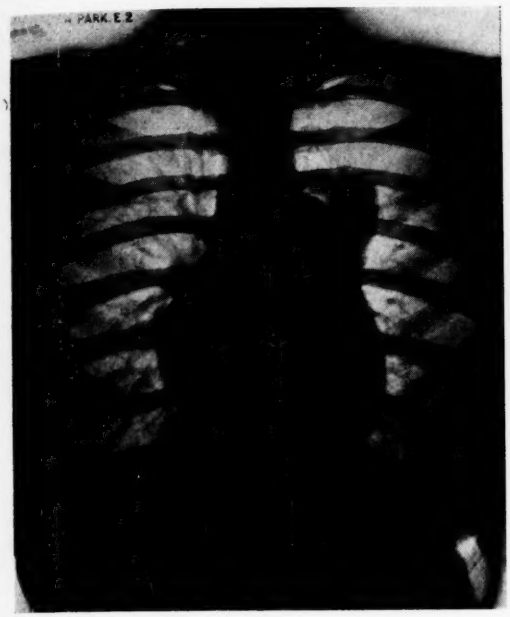

FIG. 292.—Anteroposterior X-ray picture of the chest showing an unusual shadow lying on pulmonary artery, and a 'globoid' type of heart outline.

AT OPERATION (Aug. 7, 1941).—At the Thoracic Unit, Harefield. Anæsthetic by Dr. Parry Brown—evipan, intratracheal gas-oxygen, followed by cyclopropane with controlled respiration.

Left submammary incision. The chest entered along the 4th interspace anteriorly, with division of the 4th costal cartilage.

The first glimpse inside the chest was remarkable for the appearance of a dark red triangular mass moving back and forwards in the mediastinum (*Fig.* 295). This was the uncovered auricle, which protruded in a tongue-like manner through an opening in the pericardium with each pulsation of the heart-beat. The lung lay collapsed and free from adhesions in the paravertebral gutter; it was normal in shape and in the arrangement and distribution of its lobes.

Lying on the lung was a cyst about the size of a bantam's egg, with a flat pedicle which arose from the left pulmonary artery. The pericardium was absent over a limited area, and when the cyst was lifted upwards it was seen that with its pedicle it accurately filled in the deficiency in the mediastinum. The cyst proper fitted into the superior mediastinum with two small projecting tags or ears lying flush with the upper border. The pedicle and slight downward flap fitted over the auricle and presumably this was the position maintained in life until the induction of the pneumothorax.

The left pulmonary artery was unduly large, with a curve whose convexity faced upwards, forwards, and outwards. Across this vessel at the point where it would normally be covered by pericardial reflection, the attachment of the cyst united in the form of a band about 2 or 3 mm. in thickness. No communication of any part of the cyst attachment with the bronchial tree could be established.

The most obvious part of the pericardial deficiency was above the level of the pulmonary artery in the region of the superior mediastinum, and through this opening the hand was gently passed into the right

FIG. 293.—Lateral picture of *Fig.* 292.

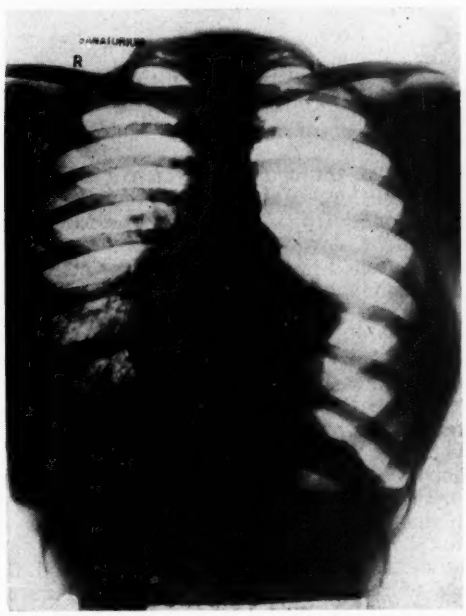

FIG. 294.—A radiograph taken after induction of left-sided pneumothorax. Air is also seen in the pericardium.

side of the pericardial sac beyond which parts of the inner aspect of the right chest wall could be palpated. There was no communication between the pericardium and the right pleural cavity. The lower part of the left side of the pericardium was intact and this prevented the ventricles from being dislodged into the left pleural sac. Actually during manipulation the heart

did become displaced from the pericardium, but it was easily put back without causing any alteration in the rhythm or force of the heart-beat.

The boundary of the pericardial deficiency was defined as follows: Superiorly it formed a curved line in the mediastinum only an inch or so below the level of the pleural dome. Anteriorly the edge descended vertically just behind the line of the

FIG. 295.—Appearance inside the chest, showing the deficiency in the left pericardium and the cyst which has fallen away from it.

phrenic nerve, the course of which it followed until it reached the level of the auriculo-ventricular septum. Here the pericardial boundary passed backwards with a slight downward curve and the edge had a definite rolled appearance. This inferior limit terminated in the lowest part of the cyst pedicle as it was reflected over the pulmonary artery. The posterior margin was ill-defined in its upper part as it consisted of a continuous reflection of the pleura on to the upper and posterior part of the pericardium. Lower down the attachment of the cyst across the enlarged pulmonary artery completed the boundary.

The base of the cyst was clamped close to the pulmonary artery, and this caused a bout of coughing. The cyst was removed and the base secured by a few transfixing mattress sutures. The chest was closed without drainage and air was aspirated.

PROGRESS.—Convalescence was uneventful, though a trace of fluid developed in the left pleural cavity and in the pericardium. The patient returned home four weeks after the operation. Her later progress has been satisfactory and she has remained free from symptoms. Radiological investigation, however, showed that the original superior mediastinal shadow has persisted, though it is smaller than before the operation. It is felt that the enlarged and curved pulmonary artery is responsible for this appearance, and it is now thought that it was the shadow of this artery rather than that of the cyst which accounted for most of the original radiological abnormality.

PATHOLOGY (Dr. Merlin Pryce).—"The cyst measures 6·5 × 3·5 × 2·5 cm. and is flattened at the cut end while being bulbous towards the other pole (*Fig.* 296).

"In most parts it is thin-walled, but thicker and botyroid on one side (the lateral or pulmonary face). This part on section is 'polycystic' in character. The cyst contains viscid yellow fluid.

"The chief and subsidiary cysts are lined by ciliated columnar epithelium. In the wall of the main 'cyst' some strands of muscles and masses of cartilage (3 in the cut section) are apparent, also secretory glands.

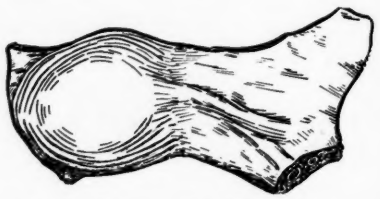

FIG. 296.—Excised cyst, showing divided bronchial elements at the pedicle. (× ½.)

"Evidence of chronic inflammation is given by lymphocytic infiltration in the wall of the main cyst: this is much more marked in the region of the smaller cysts, which contain foamy histiocytes. Alveolar spaces in the interstitial tissue contain numerous foamy histiocytes which are sometimes rendered polygonal by crowding. Epithelialization occurs in many of the alveolar spaces—the epithelium varying from cubical to tall columnar ciliated type."

The pathology was essentially one of bronchiectasis of the congenital cystic variety, and the cyst acted as part of the left pericardio-pleural membrane. It may be repeated that no communication with any other structure other than the attachment to the left pulmonary artery could be established, this in spite of the presence of bronchial elements in the pedicle. Also the existence of the cyst as a supernumerary or accessory lobe can be excluded in the ordinary sense of the term. The cyst had an association with the pericardial deficiency, and in any discussion as to the aetiology of the condition it is felt that the cyst and the deficiency should be considered together as part of the same process. Embryological study sheds some light on the possible origin of this unusual condition.

EMBRYOLOGY

The first evidence of the pericardium is the mesodermal thickening at the head end of the minute 1¼-mm. oval plate and this tissue rapidly canalizes to form the intra-embryonic cœlom. Posteriorly the cavity thus formed splits outwards to unite with the extra-embryonic cœlom, but anteriorly (and over the future pericardium) the tube remains intact as an inverted U. The posterior free arms of the U open into the extra-embryonic cœlom, and the vertical arms represent the pericardio-peritoneal channels, which will become in due course the pleural cavities. The crossbar of the U over the head end is the future pericardium.

In a short space of time the rapid growth of the head end of the embryonic plate carries the pericardium well forward, and as the dorsal aspeet moves more quickly than the ventral

surface the head of the embryo becomes bent over and the enlarging pericardial cavity in the 1·5-mm. embryo sweeps ventrally and backwards to lie under the fore-gut at the site of the primitive pharynx. This also implies that the pleural canals connecting the pericardium with the peritoneum no longer run in a straight line fore and aft: the front ends of the tubes curve forwards and ventrally to reach the pericardium.

At the tail end of the heart sac a thickening—the septum transversum—has appeared and vascular elements developing in its substance include the sinus venosus. The vessels that go to form the sinus venosus consist of the umbilical and vitelline veins and more laterally the ducts of Cuvier. The ducts of Cuvier are formed by the junction of the anterior (primitive jugular) and posterior cardinal veins and they play an important part in the closure of the pericardio-pleural channels at a later date (*Fig.* 297). At

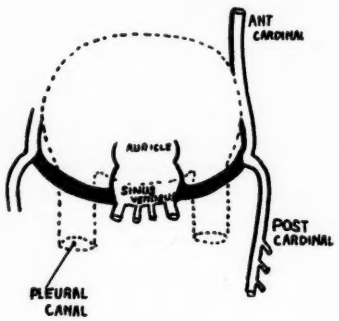

FIG. 297.—Diagram of primitive venous system. Duct of Cuvier in black.

this stage they run on the lateral side of the cœlom from the dorsal to the ventral surface of the embryo, but later as the head end of the embryo continues to develop rapidly they take a more oblique course and reach the sinus venosus, moving headwards and ventrally.

An early sign of the respiratory system is a groove in the floor of the fore-gut at the posterior end of the pharynx at the site where the sixth arches will shortly develop. By the time that the embryo has reached a 3-mm. to 4-mm. size the earliest rudiments of the lung appear as buds, and as these expand in the only direction possible to them, namely, laterally, they come into contact with the pericardio-peritoneal channels. The lungs grow into these future pleural sacs and bulge them outwards, but a centrifugal expansion is hindered by certain structures with which the growing lungs come in contact. Dorsally the body wall and caudally the septum transversum act as barriers, so that expansion occurs mainly in the ventral and head directions. This enlargement necessitates the formation of relative folds or constrictions at the front and back ends of the canal between peritoneum and pericardium, structures that may be designated as the pleuro-peritoneal and pleuro-pericardial membranes respectively. The 5-week embryo is at this stage about 8 mm. long.

The pleuro-pericardial area is important, and it will be noted that the duct of Cuvier is in lateral relation to the canal at this stage (*Figs.* 298, 299). The duct lies at the head end of the

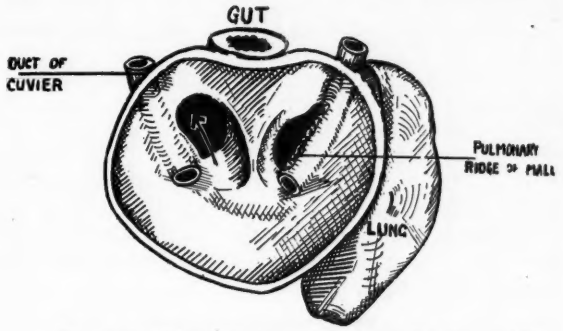

FIG. 298.—View into the embryonic pericardium with the anterior wall cut away. Arrow lies in the right pleuro-pericardial channel (iter venosum).

developing lung bud and is separated from the fore-gut elements by the cœlom; it projects into the pericardial sac to form a curved ridge on which a papillary projection (pulmonary ridge of Mall) develops. The opening between the pleura and pericardium has been given the name of the foramen or iter venosum of Lockwood, and the final closure of this foramen will lead to the isolation and separation of the two cœlomic elements.

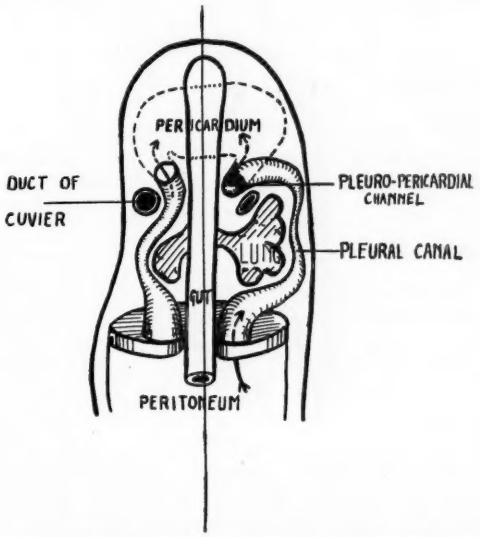

FIG. 299.—Diagram to illustrate relationship of pleural canals to the ducts of Cuvier at different stages of development. On the left the lung bud lies internal and behind the duct; on the right the growing lung has pushed its way lateral to the duct.

The next point to consider is the direction and extent of the expansion of the developing lung. To grow forwards the lung has to pass behind the duct of Cuvier before it can expand adequately; it would be impossible to the future lung apex to force its way mesially to the

duct unless the duct itself showed some abnormality. Consequently the lung tissue forming to the head end of the main bronchus lies lateral to the duct of Cuvier, which becomes driven inwards and ventrally against the pericardio-pleural canal or iter venosum. Finally by a process of elongation and pressure the iter is closed and the pleural sac separated from the pericardium (6-week embryo 12 mm. long).

During the closure of the iter venosum the pulmonary ridges which lie over the ducts of Cuvier lose their original curved course and become strained together obliquely. The ridges meet centrally at the mesodermal stump which represented the dorsal mesocardium and is continuous dorsally with the mediastinum. In this way the pericardium and pleural cavities become separated.

DISCUSSION

Complete or partial deficiency of the pericardium not uncommonly occurs alone; yet the discovery of associated congenital abnormalities is a feature of many of the recorded cases.

Keith (1912) was puzzled to explain on an anatomical basis the anomaly found in the museum specimen previously described by Curling (loc. cit.) until he was dissecting a group of malformed fœtuses, two of which displayed the condition. From a study of these specimens he concluded that the anomaly resulted from the lung bud growing within and expanding the communication between the pericardium and

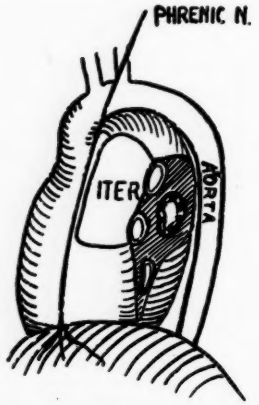

FIG. 300.—Diagram to illustrate the position of the iter venosum of Lockwood. Heart viewed from the left. The phrenic nerve is the anterior boundary. The pulmonary artery and superior vein lie posterior. (*After Keith.*)

pleura, for this communication lies immediately ventral to the point at which the lung bud appears (*Fig.* 300). He pictured the condition as a dilatation of the pleuro-pericardial foramen, a foramen which in the ordinary way closes completely, and in a later work (Keith, 1933) refers to it as a "patency of the iter venosum of Lockwood". Keith's contention as to the mechanism of the production of partial pericardial deficiency was challenged by Risel in 1912 and by McGarry in 1914. Risel observed 3 examples in newly-born infants, all of them showing multiple abnormalities which included in every case a retromediastinal bulge of peritoneum and a diaphragmatic hernia on the left side. The pericardium was partially lacking on the left side in every instance and on the right side also in one of them. He ascribed the primary cause to a persistence of the neurenteric canal. McGarry attributed the abnormalities discovered in his case, which included besides the pericardial deficiency a ventral hernia, left inguinal hernia, double femoral herniæ, and a malposition of the colon with a free mesentery, to a general disturbance of the developing cœlom. He assumes that at some stage in the course of its development the cœlom may be sensitive to injury, and attributes the almost universal occurrence of partial pericardial deficiency on the left side to the asymmetry of the liver which, in combination with the rotation of the organ during development, places a greater tension on the left pulmonary ridge. These hypotheses are both of a general character and have been based upon limited findings in a small number of cases. They are satisfactory in so far as they explain the particular character of the associated lesions, but are not applicable to the findings of other workers in which pericardial deficiency was found alone or in combination with other defects unrelated to the cœlom. Moreover, Risel's argument, in particular, takes no account of the preponderance of left-sided lesions. A right pericardial deficiency has only been recorded on four occasions in man: in three of these (Risel, 1912; Beck, 1931; and Chiodin, 1932) the abnormality was also accompanied by a left-sided deficiency, while in Egbert and Little's case (1935), which is unique, the left half of the pericardium was intact, but the right half was almost entirely missing, with the heart and right lung occupying the same serous cavity.

It is clear, therefore, that a working hypothesis must not only explain how the pericardium comes to be lacking, but must also show why its occurrence on the left side is so preponderant. Keith supplies a partial answer to the first point and his description of the foramen as a patency of the iter venosum is probably correct. It is difficult, however, to agree with him in placing the main responsibility upon the developing lung bud, and in order to envisage both how the iter venosum remains patent and why it occurs usually on the left side it is necessary to turn to the "vascular atrophy theory" put forward by Perna (1909) and Plaut (1913). These authors maintain that if the normal atrophy of the left common cardinal vein was premature then an incomplete development of the associated pleuro-pericardial membrane might result from a deficient blood-supply. When the structures which develop from the duct of Cuvier are considered it will be seen that on the right side its persistence as the wide-bore superior vena cava will ensure that the iter

venosum on that side will close and only exceptionally will it permit a patency of the pleuropericardial canal. On the left side, however, atrophy of the duct is the normal procedure. Part of the superior intercostal vein is its first definite representative: it then dwindles to a remnant which lies in the vestigial fold of Marshall, stretching between the pulmonary artery and the superior pulmonary vein, to end as the oblique vein of Marshall, a variable structure

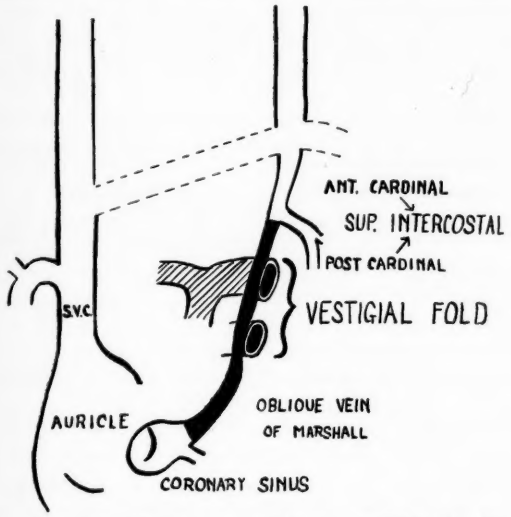

FIG. 301.—Diagram to depict the evolution in the adult of the venous elements. Left duct of Cuvier is represented in black.

which lies over the posterior aspect of the left auricle and enters the coronary sinus (*Fig.* 301). As Keith has pointed out, a failure of the iter venosum to close will result in the adult in a communication between the pericardial and pleural cavities ventral (anterior) to the remnant of the duct of Cuvier and will be limited by the extent to which the pericardium has developed at the time at which closure fails to take place. Thus, the size of the communication between pericardium and pleura will be determined by the stage of development reached when the left duct of Cuvier atrophies. An important finding is that of Niccoli (1929), who noted an associated absence of the coronary sinus and therefore gives anatomical support to the above hypothesis.

In view of the intimate relationship which exists between the lung bud and the duct of Cuvier, it would be possible for an early atrophy of the duct to lead to a disturbance of the development of the lung bud on the same side. In some of the published accounts this was found to be the case. For example, Watt (1931) found that in his case the left lung had four lobes, while Chiodin (1932) found a right lung divided by fissures into "pseudo lobes". Again, Risel (1912) described pulmonary abnormalities in two of his cases. In the course of its development the duct of Cuvier comes to lie at the head end of the growing lung bud, which in order to grow forwards and expand has to pass behind the duct. It would be difficult for the future lung apex to force its way through to the mesial side of the duct unless the duct itself were abnormal. Premature atrophy might enable this to occur. If the portion of lung bud so isolated continued to develop normally the result would be an extra lobe and one that would be ideally placed to pass through the opening of the pleuropericardial canal and come to lie within the pericardial cavity as has been described. If, on the other hand, normal development of the migratory part of the lung ceased at this point it would be well in keeping with current theories regarding the aetiology of lung cysts (Sellors, 1937) to imagine that the outcome would be a solitary cyst composed of bronchial elements and of approximately the same size as the opening into the pericardium—in other words, a cyst having precisely the characteristics and disposition of the one which was found in our patient.

A discussion of this condition would be incomplete without reference to the function of the pericardium and the significance of its absence. There have been several series of experiments conducted upon animals which have a bearing upon this point. Kuno (1915) found that opening the pericardium in dogs allowed of a larger output of the left ventricle on account of increased diastolic excursion. Parlavecchio (1909) concluded that the chief consequence of the operation was hypertrophy of the left ventricle. Carleton (1929) found that removal of the pericardium caused no alteration in heart rate or blood-pressure in cats. Radiographs were taken, and these showed that the cardiac shadow was permanently enlarged owing to dilatation and not to hypertrophy. He concluded that a restraining action is exerted by the pericardium, a restraint which is relative and not absolute, and thus disagreed with Barnard (1898), who maintained that the pericardium is an inextensible membrane.

A study of the literature indicates that in the human subject absence of the pericardium is without significance. Barsoum (1935) alone attributed the cardiac dilatation and insufficiency from which his patient died to an absence of the pericardium, which was almost total, while in Boxall's case (1887) a partial deficiency was responsible for a unique complication of the puerperium in a 28-year-old woman in whom three days after delivery the heart herniated through the foramen and thus caused death. Grant (1926) has investigated the literature with a view to determining the effect of pericardial deficiency upon heart size, and concluded that although the pericardium might support a dilating heart in such abnormal circumstances as asphyxia by drowning, it plays no part in preventing overdistension, and its absence is not a factor in causing cardiac enlargement.

A noticeable feature of many of the cases reported in the literature is the accidental discovery

of this abnormality at autopsy on men of moderately advanced age who have led strenuous lives and who have died of a totally unrelated disease.

It must be emphasized, however, that the presence of such an abnormality does increase the dangers attaching to certain respiratory diseases. Air or fluid in the pleural cavity on the same side as the pericardial deficiency might gain access to the pericardial cavity and if occurring in a tuberculous person would hamper treatment by pneumothorax as in Dahl's case already cited. It is, however, in infection that that the danger is greatest. Southworth and Stevenson (1938) found in the cases cited by them that pneumonia had caused death in 19 per cent, while in 27 per cent there was evidence of fresh pleuropericarditis. Should empyema occur, the risk to life is further increased.

SUMMARY

A case of partial deficiency of the pericardium associated with a solitary bronchogenic cyst, both on the left side, occurring in a girl of 19, is described. This is the eightieth case of pericardial deficiency to be placed on record; the third to be recognized during life; and the only one carefully studied at operation.

The symptoms of which this patient complained can be attributed to the lung cyst, which was removed. An uneventful recovery followed, and good health has been enjoyed since operation.

The literature relating to pericardial deficiency is shortly reviewed.

An account of the development of the pericardium and lung is given, and a possible mechanism for the occurrence of absence of pericardium is discussed in the light of previous theories which have been propounded. It is contended that the association of a lung cyst with a partial left-sided deficiency of pericardium lends weight to the view that the latter defect is the result of abnormal development with premature atrophy of the left duct of Cuvier.

The name given to this condition by Keith, *patency of the iter venosum*, is believed to be accurate and it possesses the added advantage of according well with the nomenclature in current use for other developmental abnormalities of the cardiovascular system.

APPENDIX

R. L. Moore in 1925 computed the total number of recorded cases of pericardial deficiency at 64. The 13 references marked with an asterisk contain the additional 15 case reports that have been found in the literature since the publication of Moore's paper.

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ACUTE APPENDICITIS AND THE APPENDIX MASS

By A. G. McPHERSON and J. B. KINMONTH

This paper analyses 730 cases of acute appendicitis treated in St. Thomas's Hospital in the period 1937–1942 inclusive. In general, we have considered the cases under the same grouping as two similar analyses of St. Thomas's Hospital cases made in 1903 and 1932 (Wallace and Sargant, 1904; Sworn and Fitzgibbon, 1932). Interesting comparison over this 40-year period is thus possible.

This series includes only definite cases of acute appendicitis, all doubtful cases have been excluded. In cases submitted to early operation clear naked-eye evidence of acute inflammation was the criterion taken, microscopical evidence alone of inflammation was not accepted. In the cases of masses where immediate operation was withheld, only those cases in which there seemed to be no reasonable doubt as to the diagnosis have been accepted. The majority of the cases in this group later submitted to interval operation showed clear evidence of previous appendicitis.

Explanation of Grouping.—The cases have been divided into three groups: (1) Simple appendicitis; (2) Appendicitis with diffuse peritonitis; and (3) Appendicitis with mass; and it is necessary to define what we mean by these terms. By simple appendicitis we mean that disease is limited to the appendix or only a localized peritonitis has occurred. Appendicitis with diffuse peritonitis is a diagnosis arrived at in part from operative findings, but since it is frequently impossible to tell the precise extent of peritonitis at operation without a dangerously extensive exploration, pre-operative physical signs such as rapid pulse, diffuse tenderness, and extensive rigidity have also been taken into account.

We mean by appendicitis with mass, a clinical condition where in addition to the signs and symptoms of appendicitis, a mass in the right iliac fossa has been palpable without anæsthesia. It has appeared to us from a study of the literature, that in those clinics where routine immediate operation is practised, no particular point is made of looking for a mass, whereas at this hospital where it is the practice to treat appendix masses expectantly, the surgeon takes special pains to ascertain, in cases where a mass might be expected, if one is in fact present. Since this may require prolonged gentle palpation, particularly where rigidity is present, it seems that many cases with mass will not be discovered unless the surgeon has this in mind.

There is little reference to the term 'appendix mass' in the literature and frequently it is confused with the term 'appendix abscess'. It is true that a certain number of masses will contain some pus or that they may on occasion become frank abscesses, but nevertheless the terms are not synonymous. We mean by 'mass' a palpable lump surrounding an inflamed appendix, and this certainly in the early stages, as we know from cases which have been operated on at this stage, consists of an inflammatory mass of œdematous adherent tissues, the appendix itself being surrounded and walled off by swollen and œdematous omentum, mesentery, and coils of bowel.

Management of Acute Appendicitis.—There is no general agreement as to the management of acute appendicitis, although it is agreed that early cases should be operated on. For the rest there are two schools, the immediate operation school and the expectant treatment school. Among those who advocate expectant treatment the indications are not always the same and therefore it is necessary to show which type of case has been treated conservatively in this series. In this series early cases have been treated by immediate operation. In later cases, over 48 hours, the patient's general condition has been carefully assessed and a mass looked for. The presence of a mass indicates an attempt on the part of the peritoneum to localize the infection, and if this attempt appears to be succeeding, interference has been regarded as meddlesome, and as long as there has been no evidence suggesting that localization has failed the case has been treated conservatively. It will be seen that operation has only been witheld where there was evidence of satisfactory localization, and we would emphasize that the clinical findings, rather than the length of history, decide the management. If there has been evidence suggesting a spread of infection in the peritoneum, or if the patient was admitted with frank diffuse peritonitis, operation was advised. This conservative treatment is not to be confused with the Ochsner treatment applied to cases with general peritonitis.

Treatment of 'Mass Cases'.—Cases with a mass and treated expectantly are put to bed in low Fowler's position, no sedatives, enemata, or aperients are allowed, fluids (in small amounts only) are given by mouth, and the half-hourly pulse-rate is charted. Indications for abandoning conservative treatment are as follows: rising pulse or persistently elevated pulse-rate (more than 100); increasing signs or symptoms suggesting a spread of infection; at a later stage abscess formation; and very occasionally a failure to resolve.

Conservative treatment is not advised for children, the aged, or in pregnancy, unless a very firm and localized mass is present on admission, and this is uncommon in these types.

Interval appendicectomy, three months after resolution of the mass, is recommended for those cases which have settled down satisfactorily under conservative treatment. Exception is made only for patients where age or general condition is such as to contra-indicate abdominal operation.

Treatment of Peritonitis.—Cases admitted with diffuse peritonitis are treated by early appendicectomy. If there is any delay its object is to improve the patient's condition by general means, such as intravenous saline or nasogastric suction, preparatory to operation.

GENERAL CONSIDERATIONS

Age.—In the present series ages range from 2½ to 82 years. *Fig.* 302, in which the age incidence for all cases is compared with that of the normal population, shows a preponderance of cases in the second and third decades of life.

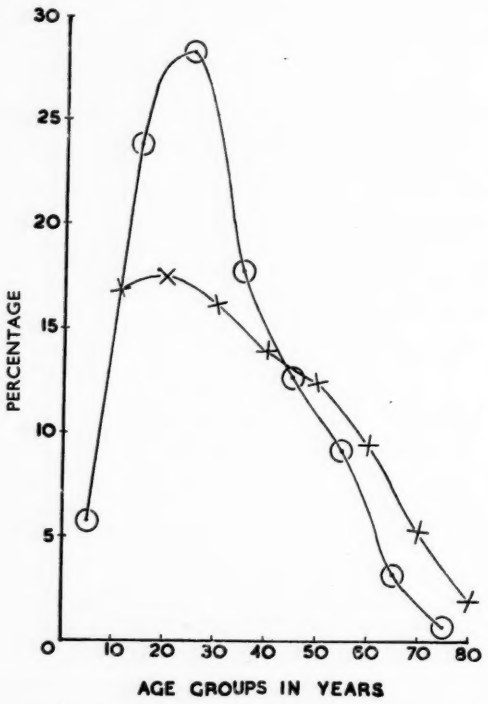

FIG. 302.—Age incidence of all forms of appendicitis compared with that of normal population. o—o, Appendicitis; ×—×, Normal population.

This confirms the findings of other authors. Most cases of peritonitis and masses also occurred in these decades. *Fig.* 303 in which simple appendicitis, peritonitis, and mass cases are compared on a basis of incidence of each per 100 cases of all types, shows: (1) a higher incidence of peritonitis at the extremes of life, and (2) a steadily increasing tendency to mass formation with age. *Fig.* 304 shows a much higher mortality-rate in older people.

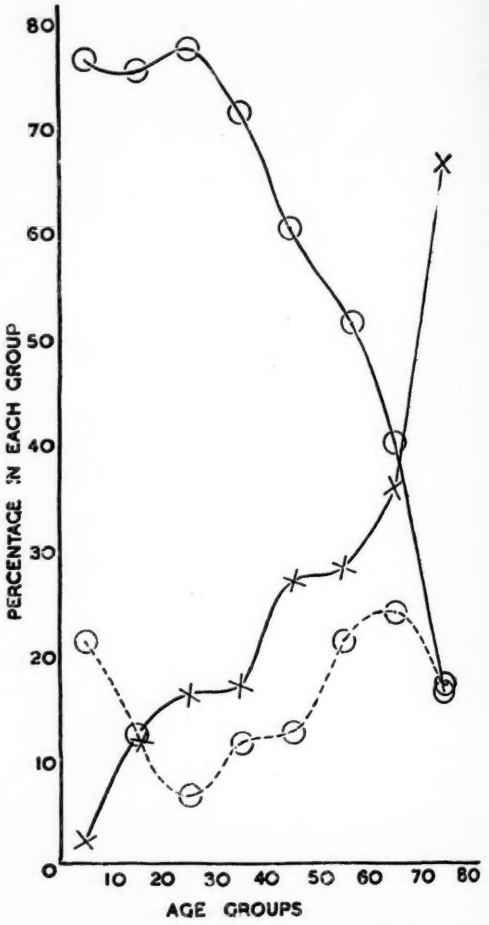

FIG. 303.—Relative incidence of different forms of appendicitis. o—o, Simple appendicitis; o---o, Appendicitis with peritonitis; ×—×, Appendix mass.

Sex.—There were 446 males and 284 females. It is important to note the preponderance of one or other sex if comparison is to be made with other series of appendicitis cases because the case mortality for the two sexes differs. Figures collected by Young and Russell (1939), show that the mortality-rate for females is about two-thirds that for males. In the present series the mortality-rate is 3·1 per cent in males and 2·5 per cent in females.

Table I.—INCIDENCE RELATED TO LENGTH OF HISTORY

	Day of Illness										
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10–14th	Over 14th
All cases	356	182	76	41	17	11	17	9	5	28	8
Simple acute appendix	336	143	17	7*	0	1	5*	1	0	0	1
Appendix with peritonitis	17	24	30	7	7	0	3	0	0	2	0
Appendix mass	3	5	29	17	10	10	9	8	5	26	7

* Two cases without mass or peritonitis, which were not operated upon, are included.

Length of History (*Table I*).—In 28 per cent of patients there was a history longer than 2 days. The average length of history for the whole series was 2·4 days. For all cases without a palpable mass it was only 1·42 days and for the mass cases 7·2 days. *Table I* shows the incidence on each day for the groups separately and combined. *Table VIII* shows that the mortality rises with increasing length of history.

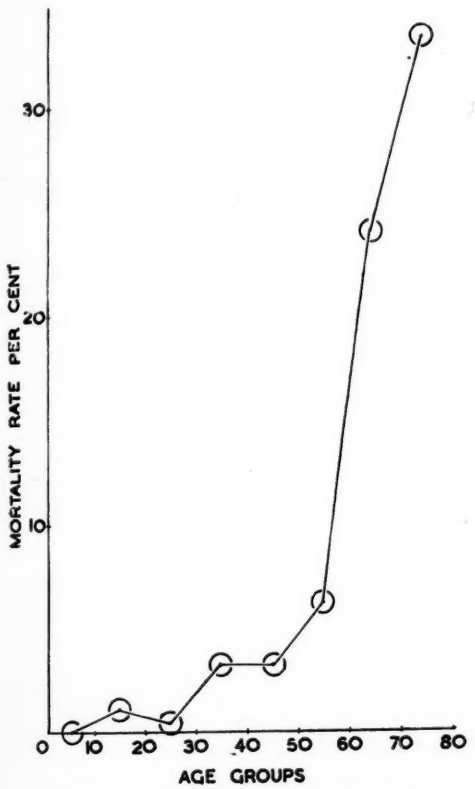

FIG. 304.—Mortality-rate in age groups (all cases).

Details of the Three Main Groups in this Series.—There were 511 cases of simple acute appendicitis, 90 with diffuse peritonitis, and 129 with a mass.

Tables II–IV analyse the groups separately, showing mortality, complications, and time in hospital.

Table II.—SIMPLE ACUTE APPENDICITIS IMMEDIATE OPERATION

DAY OF ILLNESS	NO. OF CASES	MORTALITY		COMPLICATIONS
		Cases	Per cent	
1st	336	2	0·59	26
2nd	143	3	2·15	17
3rd	17	1	5·88	2
4th	6	–	—	1
5th and over	7	—	—	1
Totals	509	6	1·17	47 (9·13 per cent)

There were 2 cases of mild acute appendicitis described on admission as 'settling' which were not operated upon. They are excluded from the above table.
Average time in hospital, 12·6 days.

Table III.—ACUTE APPENDICITIS WITH DIFFUSE PERITONITIS. IMMEDIATE OPERATION

DAY OF ILLNESS	NO. OF CASES	MORTALITY	COMPLICATIONS
1st	17	2	4
2nd	24	5	5
3rd	30	4	7
4th	7	1	1
5th and over	12	2	3
Totals	90	14 (15·6 per cent)	20 (22·2 per cent)

Average time in hospital, 30·3 days.

Simple acute appendicitis is much the largest group. Masses occur rather more commonly than diffuse peritonitis.

The mortality-rate in simple appendicitis is 1·17 per cent, in contrast to 15·6 per cent in cases with diffuse peritonitis. Only 1 case in which a mass had been felt before operation died, giving a figure of 0·8 per cent. No deaths followed interval appendicectomy.

Conservative treatment was abandoned in 31 cases and the reasons are listed in *Table V*. One case of appendix abscess, which was drained on the fourteenth day, died of reactionary hæmorrhage. Two cases appear to have been operated

Table IV.—ACUTE APPENDICITIS WITH PALPABLE MASS

SETTLED WITH EXPECTANT TREATMENT			REQUIRING EARLY OPERATION			
Day of Illness	Interval Operation	No Operation	Appendicectomy	Drainage only	Interval Operation	No Interval Operation
1st	2	0	1	1	0	1
2nd	13	3	5	0	0	0
3rd	11	8	6	1	1	0
4th	6	6	1	0	0	0
5th and over	30	19	11	5	3	1
Totals	62	36	24	7	4	2

Total all cases, 129 with 1 death (0·8 per cent); this followed simple drainage of a fifth-day case.
Average time in hospital, 32·9 days. Complication rate, 8·2 per cent.

upon without clear indication: in 1 case the clinical mass disappeared rapidly and operation was performed on the eleventh day; appendicectomy proved very difficult owing to firm adhesions and the cæcum was torn during the manipulations. In the second case, which was operated upon on admission, without definite indication, a very firm mass was found and cœliotomy only was performed.

Table V.—Indications for Operation in Cases with Palpable Mass on Admission

	Cases
Diffuse peritonitis, immediate operation	6
Deterioration during observation period	5
Abscess present	1
Abscess developed during observation period	7
Recurrent attack during 'interval'	4
Age (child)	1
Uncertainty as to nature of mass	5
None noted	2
Total	31

Complications.—The complication rate is highest, as would be expected, in the diffuse peritonitis group. It is to be noted that it is lowest in the palpable mass group, including those which came to operation. We have excluded from *Tables II–IV* cases where healing by first intention was not obtained through suppuration in the wound, but without other complications. These cases appear to be omitted by other authors and are of frequent occurrence.

Table VI shows details of the complications occurring in the various groups. Many of these are chest complications, e.g., atelectasis and bronchitis, and are not directly related to the appendicular lesion.

Table VI.—Non-fatal Complications of the Three Main Groups

	Simple Appendicitis	Appendicitis with Peritonitis	Appendicitis with Mass	All Cases
Hæmatoma	3	—	—	3
Residual abscess	2	3	1	6
Pelvic Abscess	7	7	3	17
Fæcal fistula	—	1	—	1
Paralytic ileus	—	3	—	3
Acute obstruction	1	1	—	2
Burst abdomen	—	—	1	1
Bronchitis	7	1	—	8
Atelectasis	18	—	3	21
Pneumonia	4	2	—	6
Pulmonary infarct	3	1	2	6
Femoral thrombosis	1	1	—	2
Delirium tremens	1	—	—	1
Total cases	511	90	129	730
Total complications	47 (9·2 per cent)	20 (22·2 per cent)	10 (7·8 per cent)	77 (10·5 per cent)

Morbidity, and the Mortality-rate of Mass Cases.—The time spent in hospital averaged 12·6 days in simple appendicitis, 30·3 days in general peritonitis, and 32·9 days in the mass cases returning for interval appendicectomy.

One criticism of the practice of delaying operation in selected cases is that a longer stay in hospital is required.

Another is that a number of patients default and do not return for interval appendicectomy.

The percentage who did not have interval operation in this series is unquestionably a large one, but we feel that the number is larger than would occur in normal times, since the majority of these cases have occurred during the war. Under the present circumstances it is more difficult to keep in touch with patients and more difficult to persuade them to return to hospital when they have felt fit and perhaps been doing remunerative or important war work.

In spite of these criticisms we feel that the mortality is the important deciding factor, and it is significant that 1 only of the mass cases died, and this followed operation for appendix abscess. The mortality-rate for masses is the lowest of the three groups (including simple acute appendicitis), and this despite the longer average history—a factor usually agreed to raise the mortality. Would it be still lower if immediate operation was the rule? A categorical statistical answer is impossible. One cannot get a series of masses treated by immediate operation for comparison, because surgeons practising such a method do not recognize or classify such cases in a separate category.

Nevertheless, it seems unlikely that immediate operation on these cases would produce a lower mortality-rate than for simple acute appendicitis. Further, on general surgical grounds it does not appear to us to be a sound policy to interfere with an infective process in the peritoneum where there is evidence that localization by natural processes has already been achieved.

Observations on the Fall in Mortality-rate in 40 Years.—It is interesting to follow the fall in mortality of acute appendicitis in the last 40 years as shown in *Table VII*, and to postulate causes for this.

Table VII.—Comparison with Previous St. Thomas's Hospital Figures

Series *A*. 1894–1903— 433 cases with 182 deaths (42 per cent)
Series *B*. 1920–1929—1755 cases with 86 deaths (4·9 per cent)
Series *C*. 1937–1942— 730 cases with 21 deaths (2·9 per cent)

I. Acute appendicitis without peritonitis :—
A. 13 cases 0 deaths
B. 1340 cases 23 deaths (1·17 per cent)
C. 509 cases 6 deaths (1·17 per cent) (operated cases only)

II. Acute appendicitis with diffuse peritonitis :—
A. 166 cases 143 deaths (86·0 per cent)
B. 226 cases 48 deaths (21·1 per cent)
C. 90 cases 14 deaths (15·6 per cent)

III. Cases with clinically palpable mass :—
A. 254 cases 39 deaths (15·3 per cent)
B. 189 cases 15 deaths (7·9 per cent)
C. 129 cases 1 death (0·8 per cent)

In the earliest series (Wallace and Sargent, 1904) appendicitis was treated in medical wards and the surgeon was called in at a later stage.

Of the patients in Sworn and Fitzgibbon's series (1932) 48 per cent had a history longer than 48 hours, compared with 28 per cent in this series.

Table VIII shows the increasing mortality of cases after the first day. The time factor is, in our opinion, one of the greatest, if not the greatest factor in the lowered mortality-rate.

compared with 24 per cent in the present series, many more of these being operated on at once. It is amongst these early operation cases that the deaths occur.

Analysis of Deaths.—*Table IX* shows the causes of death in the 21 fatal cases. Diffuse peritonitis, as would be expected, accounts for the largest number. Other causes are individually few.

Table IX.—CAUSES OF DEATH

Causes of Death	*No. of Cases*
Diffuse peritonitis	12
Subphrenic abscess—general peritonitis	1
Bronchopneumonia	1
Hæmorrhage and shock	2
Secondary hæmorrhage	1
Pulmonary embolus	1
Paralytic ileus—without peritonitis	1
Gas gangrene of wound	1
Hypertensive cardiac failure	1

Two cases died of shock and hæmorrhage and these deaths are theoretically avoidable.

Table VIII.—MORTALITY RATE IN RELATION TO LENGTH OF HISTORY

DURATION OF SYMPTOMS ON ADMISSION	DAYS								
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th
No. of cases	356	171	75	29	17	11	15	9	5
No. of deaths	4	8	5	1	2	0	1	0	0
Mortality rate per cent	1·1	4·7	6·7	3·4	11·8	—	6·7	—	—

A point which may be of importance, and one that has been frequently emphasized in the past, is the danger of aperients. We have no accurate data as to the number of cases which had been given purgatives before being sent to hospital, but it is our impression that the number is small and that the general public and the practitioner are both more fully aware nowadays of the danger of giving purgatives for undiagnosed abdominal pain.

Intravenous drip saline and continuous naso-gastric suction have been fully employed in the present series in cases with peritonitis, and we consider these of great value.

Although some of our late cases have been given sulphonamides, these drugs have only rarely been given intraperitoneally and we are doubtful if they have had any influence on the mortality figures in this series.

Changes in anæsthetic technique and the more frequent use of local anæsthesia in the worse cases may also have helped to lower the mortality.

The low mortality of cases with a mass is remarkable, and the drop in mortality in this group as compared with the figure of Sworn and Fitzgibbon (1932) is very much larger than the drop in the earlier groups. All the above-mentioned factors influence this, but, perhaps the most important factors are the improvement in the selection and the technique of management of cases for conservative treatment. A greater proportion of the mass cases in Sworn and Fitzgibbon's series had early operation (38 per cent)

Of the 8 cases where a previously unperforated appendix was ruptured on removal, 2 died, one of a spreading gas infection of the abdominal wall and the other of a pulmonary embolus. Both of these cases had short histories of under 24 hours and showed no diffuse peritonitis at operation and belong to a type of case normally carrying a good prognosis.

No case of simple appendicitis or appendicitis with mass died of diffuse peritonitis, though this was the cause of death in practically all those of the peritonitis group.

The average age of the fatal cases was 46·3 years, much higher than the average of the whole group. Eight patients were aged 60 or over. This conclusively demonstrates that the prognosis is much worse in the aged.

Comparison with other Series in the Literature.—It is not possible to make any strict comparison between the figures in different series, and it may indeed be misleading. There are far too many variable factors involved.

The criteria of diagnosis are often different—in some series milder cases are included on microscopical evidence of acute inflammation only, or 'subacute' or chronic cases are accepted.

Different sex composition of series also invalidates comparison.

It is well known that collective figures for an institution should not be compared with those for individual surgeons without allowance being made. Other things being equal, better figures are naturally to be expected from the individual.

A series in which a high proportion of peritonitis cases occurs cannot be compared with one with a smaller proportion of these cases.

Comparison of the various types of acute appendicitis is even more difficult as the methods of grouping adopted by different authors varies enormously. This is especially so with 'mass' cases, as such a subdivision is very rarely made. Many authors subdivide their cases into numerous types: e.g., peritonitis—'local', 'flank', 'diffuse', 'spreading', 'general', and such terms occur frequently in the literature. It is difficult to understand how the separate entity of these groups can be clearly established.

Comparison of subgroups also means comparison of smaller numbers of cases, so that errors of chance play a larger part.

Therefore, we have not attempted any comparison of subgroups of appendicitis, but include in *Table X* some figures from the literature to which fewest of the above invalidating objections apply.

Table X.—Mortality Rate for Acute Appendicitis (all types) in Other Series in the Literature

Author	Date	No. of Cases	Mortality Rate Per cent	Remarks
Nuttall	1935	617	2·6	Immediate operation
Holman	1938	1,200	4·8	Immediate operation
Stafford and Sprong	1940	1,317	3·6	Immediate operation
Barrow and Ochsner	1940	1,039	5·3	Some peritonitis cases treated conservatively
Miller et al.	1940	656	5·4	Expectant treatment for masses. All patients children
Mason et al.	1941	1,163	1·5	Expectant treatment for masses. Very few peritonitis cases.
Present series	1943	730	2·9	Expectant treatment for masses

It is interesting to note the figures of Miller and his co-authors (1940). Their patients were all children, the same grouping is applied as we have used in our series, and the same regimen of expectant treatment adopted for masses—an unusual practise where children are concerned, but one which their low mortality figure of 3·1 per cent for such cases appears to justify.

In reviewing the recent literature relating to the treatment of acute appendicitis we have noticed that nearly all authors, however keen protagonists they may be of the immediate operation policy, make some reservations. There seems to be a feeling that there are cases better treated conservatively and very often it is expressed by a remark about late cases with a palpable mass (felt clinically), or it may be in a word of advice about dealing with an appendix mass found at laparotomy. The 'mass' does not find much place in their statistics, however, and we feel that it is more common than is generally realized and that searching for it a little more carefully and treating it expectantly well repays the surgeon.

SUMMARY

A total of 730 cases of acute appendicitis treated at St. Thomas's Hospital have been analysed. Particular attention has been drawn to acute appendicitis with mass formation.

Conservative management of appendix masses is recommended and rules for selection of cases for expectant treatment are given. The figures quoted justify this conclusion.

The effects of age, sex, and length of history on mortality are discussed.

The mortality-rate for the whole series is 2·9 per cent, for simple acute appendicitis 1·17 per cent, for peritonitis cases 15·5 per cent, and for masses 0·8 per cent.

Complications and length of stay in hospital are discussed.

A comparison is made with previous series treated at St. Thomas's Hospital during the last 40 years.

Analysis is made of the individual causes of death.

The literature has been reviewed and the figures of a few of the more comparable recent series have been tabulated.

Our thanks are due to the Surgeons of St. Thomas's Hospital for permission to publish the results of these investigations.

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STENOSIS OF THE SMALL INTESTINE FROM THE CICATRIZATION OF ISOLATED INFLAMMATORY LESIONS

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STENOSIS of the small intestine would be symptomless until the lumen of the bowel were so narrowed as to interfere with the passage of the intestinal contents. As the contents of the small intestine are fluid considerable narrowing would occur before symptoms were caused, and it has been justly estimated that the lumen of the bowel could be narrowed to the diameter of a lead pencil without causing symptoms. An inflammatory lesion could not produce stenosis until some considerable time had elapsed from its inception.

Whether an inflammatory lesion of the small intestine could produce symptoms apart from those of stenosis is debatable. The exudate from an isolated inflammatory lesion would be negligible, and an isolated ulcer in the small intestine would probably be symptomless. Some writers have, however, attempted to describe clinical syndromes for ulcers of the small intestine on the same plan as the clinical syndrome for gastric and duodenal ulcers. Smith (1935) states that although no typical group of symptoms has been encountered, an abstract of the literature makes possible a collection of symptoms found in proven cases of jejunal ulcers. Pain in the epigastrium or left upper abdomen made worse by eating; pain which is non-radiating, appearing at no appointed time as regards meals and of a dull boring character; pain unrelieved by the taking of food or alkaline powders; pain with intervals of freedom of two or three months; occasional nausea and an indefinite area of tenderness in the left upper abdomen make up this syndrome. It is unlikely that a laparotomy would be performed for such a syndrone and the validity of this syndrome remains undecided at present. However this may be, it is certain that most cases are symptomless until there is mechanical obstruction to the flow of the intestinal contents. At this stage the patient would experience recurrent attacks of circum-umbilical abdominal pain, and observation would demonstrate visible peristalsis of the ladder pattern associated with these bouts of pain. Such a clinical picture is unmistakable, and calls insistently for relief by a surgical operation. Vomiting might or might not be present, but would be more marked the higher the obstruction in the intestine. An X-ray picture taken after a barium meal would show dilated coils of small bowel containing gas, and sharp horizontal fluid levels between the gas and the barium meal. The clinician would naturally consider the question of the cause of the stenosis, but in most cases no diagnosis in regard to cause could be made before operation.

At laparotomy there would be found dilated coils of small bowel and contracted empty coils of bowel and the obstruction would be located at the junction of the dilated bowel with the contracted bowel. Inspection of the bowel might enable an opinion to be given as to the cause of the stenosis. A tuberculous ulcer causes a scar which spreads circumferentially around the bowel from its antimesenteric border. In some cases the stricture of the bowel is associated with other scars of this nature on the walls of the bowel. Close inspection of the bowel might reveal tubercles on the peritoneal aspect of the scar and there might be caseating glands in the mesentery. In other cases an annular stricture is present, which must have resulted from the scarring of an inflammatory lesion. Whether the lesion be of tuberculous origin, or the non-specific granuloma, first described by Crohn (1932), would be impossible to decide from an external inspection of the bowel. Crohn's disease would be more likely if there were involvement of an appreciable segment of the bowel, but it must be conceded that Crohn's disease might not involve an appreciable segment of the bowel. Attention has been drawn in the literature to the diagnostic significance of the 'string sign' of Kantor (1934) where a trickle of barium along a stricture of the small intestine is diagnostic of the presence of Crohn's disease. A malignant growth could cause an annular stricture of the bowel, but there is usually considerable body to such a stricture from the presence of tumour tissue. Some cases might, however, simulate an inflammatory stricture. Operative relief of a stricture of the intestine could be obtained by short-circuiting the stricture by means of a lateral anastomosis, and where a stricture represents a healed scar of a past infection such treatment would cure the patient by an operation involving a minimum risk to life. The other method of treatment would be to resect the affected segment of the bowel containing the stricture with restoration of the continuity of the bowel by an end-to-end anastomosis. Where there was any possibility of the stricture being malignant such a course would be the best assurance against a possible disaster. Patients stand these surgical operations remarkably well, and convalescence is usually smooth. It is possible that with the methods of aseptic anastomosis now in vogue, even better results would be possible, with an afebrile post-operative convalescence.

When the bowel has been resected, it is possible to make a detailed examination of the

lesion. There is usually a small ulcer on the antimesenteric border of the bowel. Histological examination of the floor of the ulcer would demonstrate tuberculous granulation tissue in the case of a tuberculous lesion, provided that healing had not progressed so far as to cause replacement of the granulations by scar. In the case of Crohn's disease granulations would consist of plasma cells and young connective-tissue cells. A diagnosis of Crohn's disease would be made by exclusion; for if the lesion were tuberculous the granulations would be characteristic of tuberculosis, and if it were malignant the histological appearances would be characteristic of malignant disease.

CASE REPORTS

Case 1.—**Acute intestinal obstruction due to a tuberculous stricture of the ileum.**

F., male Singhalese, aged 50 years, was admitted to the General Hospital, Colombo, on March 12, 1936, at 10.20 a.m. on account of abdominal pain of two days' duration. The patient was deaf and a history was difficult to elicit. The pain was circumumbilical and colicky, coming on in bouts every few minutes and he had vomited once on the day of admission to hospital. He had not had a stool, nor had he passed flatus since the onset of the pain. While being questioned, the patient vomited watery brown fluid. The abdomen was moderately distended,

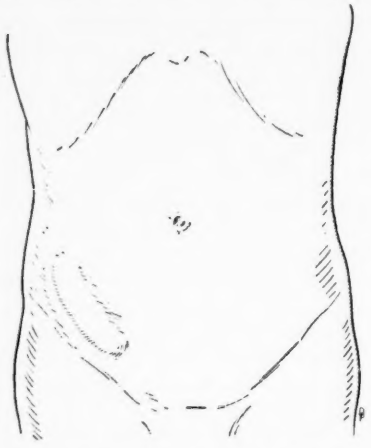

FIG. 305.—*Case* 1. Diagram showing the size of the sausage-shaped lump palpated in the abdominal cavity.

and through the abdominal wall there were clearly visible contracting coils of bowel arranged in a ladder pattern, hardening under the palpitating hand with each bout of pain. A sausage-shaped lump was palpable in the right iliac fossa lying parallel to the groin and about six inches long (*Fig.* 305). An enema was given, but the enema fluid was returned without force, and without flatus.

A diagnosis of an ileo-ileal intussusception was made, and immediate operation advised.

OPERATION.—This was carried out at 11.30 a.m. The abdomen was opened through a right paramedian incision. The peritoneal cavity was clean, there being a little excess of serous fluid in the peritoneal cavity. Contracted empty coils of small bowel were seen, with dilated loops of bowel amongst them. On exploring the right iliac fossa, there was found a distended loop of small bowel, 6 in. long, heavy with putty-like fæces. At the distal end of this loop of bowel, there was a sudden narrowing of the lumen of the bowel by a white annular stricture. The bowel beyond the stricture was empty and contracted while the bowel above the coil containing putty-like fæces was dilated. At the site of the stricture there was a white puckered scar on the antimesenteric border of the bowel with a white streak running around the bowel on each side towards the mesenteric border. There were no other scars on the intestinal walls. The unusual circumstances of finding putty-like fæces firmly impacted over 6 in. of ileum above the stricture greatly complicated the operative procedure. A short-circuit of the ileum above and below the stricture would not have relieved the affected loop of ileum of its heavy load of fæces, and it was decided that this loop must be resected. This was done, 10 in. of ileum being removed, and the continuity of the bowel re-established by an end-to-end anastomosis. The abdominal wound was closed.

The patient made a good recovery from the operation, passing flatus on the fourth post-operative day and a solid stool on the fifth. The wound healed by first intention and the patient left hospital on March 23, 1936.

FIG. 306.—*Case* 1. Photograph of transverse scar on the antimesenteric border of ileum.

THE SPECIMEN.—This shows 10 in. of ileum, 3 in. being below the stricture and 7 in. above. The mucosa shows healthy valvulæ conniventes and it is neither ulcerated nor scarred. From the peritoneal aspect there is seen a puckered scar the diameter of a lead pencil on the antimesenteric border of the bowel, with a transverse white scar passing round the bowel wall (*Fig.* 306).

Commentary.—There was in this case but a single white scar on the bowel wall, which from its position and distribution must have been produced by cicatrization of a single tuberculous ulcer of the ileum. There was found no other evidence of tuberculous infection of the abdominal cavity. The finding of solid fæces in the ileum is unusual and it is possible that this was caused by continued inspissation of the bowel contents by absorption of water by the ileum. It is known that patients with a permanent ileostomy pass semi-solid stools after some time, and a similar adaptation must have taken place above the stricture in this case.

FIG. 307.—*Case* 2. Radiograph showing condition five hours after the administration of a barium meal. The gas in the greatly dilated small intestine with the horizontal fluid levels between the gas and the barium is well shown.

Case 2.—**Subacute intestinal obstruction due to a tuberculous stricture of the jejunum.**

S. A. R. A., aged 30 years, male Singhalese, was admitted on Aug. 12, 1942, to the General Hospital, Colombo, on account of severe bouts of colicky abdominal pain. At the time of admission the bouts of pain came on every ten or fifteen minutes. These pains had been noted for the last four months, at first coming on two or three times after meals and lasting on and off for two or three hours at a time. The pains had gradually come on more and more frequently. During the height of a pain, the patient could see a lump forming in his abdomen as if the bowels had become knotted. In the last two months there had been vomiting of fresh food once a day. The patient stated that he had been operated on fifteen years ago by Dr. S. C. Paul for abdominal pain, and since that time had no trouble till the onset of the present illness.

ON EXAMINATION.—The patient was a well-nourished individual, who was perfectly comfortable till the onset of a bout of pain. The pain was circum-umbilical and at the height of the pain there was visible through the abdominal wall the ladder of small-intestine peristalsis. The coils of contracting bowel could be palpated at the height of the pains, whilst between the pains the abdomen was free from tenderness, there being no lump palpable at these times. The scar of an old paramedian abdominal incision was seen. Pulse 80 and of good volume. Although it was clear that the patient was suffering from an organic obstruction of the small intestine, in view of his good general condition it was decided to operate on the patient on the next operating morning, the interval being utilized to take an X-ray picture after the administration of a barium meal. The five-hour picture (Aug. 13) shows greatly dilated loops of small bowel filled with gas and containing barium with a clear fluid level between the gas and the barium (*Fig.* 307).

OPERATION (Aug. 14).—Under light percaine anæsthesia, the abdomen was opened through a right paramedian incision. The peritoneal cavity was clear and dry. Both dilated and contracted coils of small bowel were seen in the abdominal cavity. The dilated bowel was traced downwards and it was found

to terminate abruptly at the site of an annular stricture on the bowel wall about midway down the jejunum (*Fig.* 308). The rest of the small bowel was empty. No other lesion of the small intestine was noted, and there were no enlarged glands in the mesentery. The appendix was missing. Palpation of the stricture gave the feel of an indurated ring. On the peritoneal coat there was a white scar running transversely

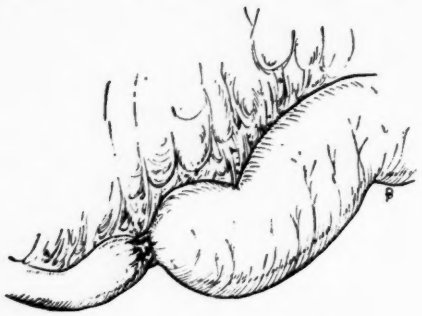

FIG. 308.—*Case* 2. Diagram showing annular stricture with transverse white scar found.

round the bowel at the site of the stricture. It was considered that the stricture was due to a healed tuberculous lesion, but it was felt that the possibility of the stricture being caused by a malignant growth could not be excluded. In view of this, it was

FIG. 309.—*Case* 2. Photograph showing puckered scar with shallow ulcer of mucosa at antimesenteric border.

decided that resection of the bowel would be preferable to short-circuiting the stricture, and accordingly 4 in. of bowel were resected, the continuity of the bowel being restored by end-to-end anastomosis. On account of marked inequality between the lumen of the dilated proximal end and that of the contracted distal end, the excess lumen on the proximal end was closed off from the antimesenteric border. Five hours after operation the patient was complaining of severe colicky abdominal pains coming on in bouts every few minutes. His pulse was 68 and of good volume. These pains continued for nearly six hours after operation, after which they suddenly disappeared when the patient passed flatus for the first time.

Five days after operation the patient passed a large solid stool and on the next day his bowels opened again. The wound healed by first intention and the patient left hospital on Sept. 4, feeling his normal self.

THE SPECIMEN (*Fig.* 309).—Viewed from the peritoneal side there is an annular stricture of the jejunum with a fibrous ring going all round the bowel. On opening the bowel there is seen an annular scar on the mucosa with a small shallow ulcer on the mesenteric border of the bowel at the site of the scar.

Case 3.—**Acute intestinal obstruction due to a tuberculous stricture of the ileum.**

J. A., male Singhalese, aged 52 years, was admitted to the General Hospital on Dec. 4, 1941, under Dr. G. S. Sinnethamby, complaining of colicky diffuse abdominal pain of seven days' duration. Vomited twice. Absolute constipation. The case was referred to the writer for examination and operation.

ON EXAMINATION.—At the time the patient was having the pains every few minutes and on each occasion the ladder pattern of visible peristalsis was clearly seen. On auscultation the turbulent sounds of exaggerated peristalsis were heard. A rectal examination revealed a bulging of the rectal wall above the prostate and in the bulging area the outline of dilated coils of bowel could be distinguished. Pulse 65, temperature 98·4°. A diagnosis of organic obstruction of the lower ileum was made and an emergency operation advised.

OPERATION.—This was done an hour later under light percaine anæsthesia. A right paramedian incision was made and the abdominal cavity opened. The peritoneum was clean and dry. The abdomen was full of dilated coils of small intestine. On exploration of the bowel, a stricture was found in the ileum, 12 in. from the ileo-cæcal valve. At this point the bowel narrowed suddenly, and the bowel below was empty and contracted, as was the large bowel (*Fig.* 310). The stricture was an annular one, having

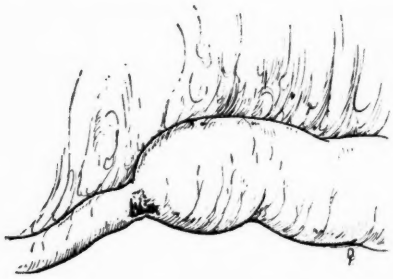

FIG. 310.—*Case* 3. Diagram showing bowel at site of stricture. The transverse white scar on the antimesenteric border of the bowel is shown in the figure.

a transverse white scar on the antimesenteric border extending round each side of the bowel. The mesentery of the small intestine was white and scarred, and there were a few light adhesions here and there, which were readily separated. No other scars were noted on the bowel walls. A lateral anastomosis between the ileum above and below the stricture was made and the abdominal wound closed. Six hours after operation the patient passed a large stool and another stool twelve hours after this.

The patient made a good recovery. Nine days after operation there was a discharge of pus from the abdominal wound, but after this healing was uninterrupted. He left hospital on Jan. 7, 1942, relieved of all symptoms.

Commentary.—In this case the clinical picture of a lower ileum obstruction was clear, and at the operation the finding of a transverse white scar at the site of the stricture made possible a definite diagnosis of tuberculous stricture. The early passage of two large stools a few hours after operation was due probably to the vigorous peristalsis promoted by the spinal anæsthetic.

Case 4.—**Chronic intestinal obstruction due to a tuberculous stricture of the jejunum relieved by gastro-enterostomy.**

G. M. A., male Singhalese, aged 30 years, was admitted to the General Hospital on June 12, 1941, complaining of colicky diffuse abdominal pain of two months' duration, and frequency of micturition for twenty days. No nausea or vomiting. He had had similar attacks of abdominal pain of one or two weeks' duration during the past one and half years.

On examination the abdomen was soft and no lump was palpable. Per rectum the prostate was of normal shape.

Urine: Cloudy, Sp. gr. 1012, Alb. +.
Deposits: Pus cells +, field full.
Cystoscopy: Bladder normal.
Radiography: No vesical calculus seen.

The circum-umbilical pains continued on and off, but as no definite lesion was discovered the patient left hospital on June 27, promising to return for further investigation.

On Nov. 11 the patient was re-admitted on account of abdominal pains of one month's duration. The pain was circum-umbilical. The pains came on in

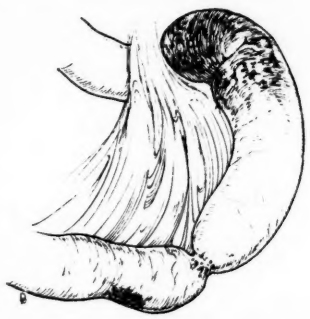

Fig. 311.—*Case* 4. Diagram showing area of induration of jejunum just beyond duodeno-jejunal flexure and the annular stricture 3 in. beyond this.

bouts and were worse after meals, coming on five minutes to half an hour after meals. No vomiting. The patient still had frequency of micturition, day 10 times, night 15 times, and a dull pain in the lower abdomen. Abdomen soft. Spleen enlarged two fingers below costal margin. On radiography after a barium meal the stomach was seen to fill well without a defect and to empty in the normal time. The duodenal cap was visualized. There was some stasis in the cæcum after twenty-four hours.

Urine: Alb. nil, occasional pus cells in deposit, culture sterile.

Examination of the central nervous system revealed no abnormality.

There appeared to be two conditions in this case —the urinary trouble and the colicky abdominal pains coming on after meals. As the abdominal pains were the more severe of the two, an operation was advised.

OPERATION.—Under light percaine anæsthesia, the abdomen was opened through a midline supra-umbilical incision. The stomach, duodenum, and gall-bladder were found to be normal. The bowel was now inspected from the duodeno-jejunal flexure. The first part of the jejunum just below the flexure was indurated and pulpy for 3 in., and 3 in. beyond this was an annular stricture of the jejunum. At this site there was a transverse white scar going all around the bowel (*Fig.* 311). There were several scars about every 9 in. of the jejunum, becoming less and less marked lower down, and not being present in the ileum or large bowel. Many of these scars were on the mesenteric border of the bowel wall and others

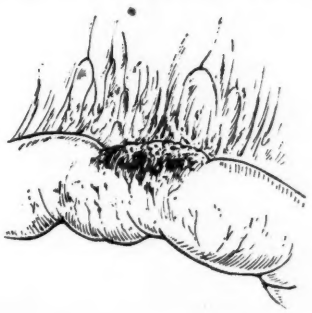

FIG. 312.—*Case* 4. Diagram showing scar in mesenteric border of jejunum which was œdematous. There were miliary tubercles on it, which are shown in the diagram.

on the antimesenteric border, these being transverse to the long axis of the bowel (*Fig.* 312). Miliary pinhead tubercles were seen on the scars in some places and the scars were œdematous. It was clear that this patient had multiple tuberculous lesions of the jejunum, and that the annular stricture within a few inches of the duodeno-jejunal flexure probably accounted for his abdominal pain. A posterior gastro-enterostomy was done between the stomach and the jejunum beyond this stricture. The patient made a good recovery from the operation. The diffuse abdominal pains were relieved after the operation, but the patient now complained of dysuria, and the pain in the hypogastrium became more noticeable.

The urine was examined for the bacillus of tuberculosis, but the test was negative.

The patient left hospital on Jan. 16, 1942.

Commentary.—In this case, the lesions in the jejunum with their multiple white œdematous scars, some of them transversely disposed on the bowel wall, and many with miliary tubercles in their scars, pointed clearly to the diagnosis of tuberculosis. The single jejunal stricture found in this case was so close to the duodenum that the simplest mode of relieving the obstruction was by a posterior gastro-enterostomy. It is very probable that the patient also had tuberculosis of one or both kidneys in view of this continued complaint of dysuria and pain in the hypogastrium with clear urine and a bladder normal on cystoscopic examination.

Case 5.—**Crohn's disease causing a stricture of the jejunum cured by duodeno-jejunostomy.**

H. K. D. M., aged 37, male Singhalese, was admitted to the General Hospital on Jan. 17, 1944, on account of great emaciation and persistent vomiting

of four months' duration. The patient had been advised to undergo a surgical operation for this condition one month ago, but had tried medical treatment instead. Two years previously he had a bout of pain lasting one week, but since then he had kept in good health and had had no symptoms till four months ago, when he began to vomit once in every three or four days. The vomiting had no relation to meals and the vomitus was a yellow bitter fluid. The vomiting had persisted up to the present time, coming on once in two to four days to twice a day. This vomiting had been accompanied by constipation, loss of appetite, and extreme emaciation.

ON EXAMINATION.—The patient was emaciated, his eyes were sunken, his abdominal skin had lost its elasticity, and the ribs were merely covered by skin. There was visible peristalsis of the gastric type, the waves moving from left to right across the upper abdomen.

An X-ray picture showed marked dilatation of the stomach and duodenum with retention of the barium meal in the duodenum up to five hours (*Fig.* 313).

OPERATION (Jan. 21).—Under local infiltration anæsthesia the abdominal cavity was opened by a

FIG. 313.—*Case* 5. Radiograph immediately after a barium meal. The stomach and the duodenum are dilated and full of barium. There is a stricture of the jejunum 1 in. from the duodeno-jejunal flexure.

right paramedian incision, 6 in. long and centred on the umbilicus. The small intestines were collapsed and empty. There was an annular stricture of the jejunum, 1 in. from the duodeno-jejunal flexure, and 2 in. beyond this was a second area of jejunum 1 in. long which was not normal. Both the affected areas were indurated, œdematous, and salmon-pink in colour. The glands of the mesentery of the affected jejunum were enlarged to the size of peas. The jejunum beyond the diseased area was anastomosed to the third part of the duodenum by a lateral anastomosis, no clamps being used (*Fig.* 314).

The patient made an uninterrupted recovery from this operation, and left hospital on March 5, being kept for this period in order that he might regain his lost weight by adequate feeding.

The diagnosis of Crohn's disease is suggested by the indurated salmon-pink areas of bowel, the second lesion being 1 in. long.

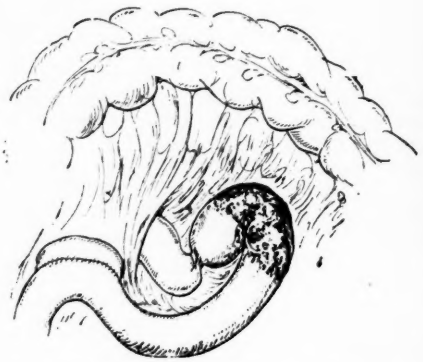

FIG. 314.—*Case* 5. Showing the affected areas of jejunum and the completed duodeno-jejunostomy.

DISCUSSION

The cases reported in this paper do not appear to be of common occurrence. Caplan and Roantree (1939) described 2 cases of stricture of the ileum in South Indians—one definitely due to tuberculosis and the other almost certainly of the same origin. They refer to a paper by Chi (1934), who was able to find records of only 11 cases of localized tuberculosis of the ileum reported up to that time. The pathology of non-tuberculous chronic inflammatory strictures of the intestine has still to be worked out fully. Idiopathic ulcers in both jejunum and ileum have been described by Brown (1924), Ebeling (1933), and Smith (1935). In the present state of our knowledge it would appear that such ulcers should be classified as cases of Crohn's disease. Kini (1941) described 3 cases of granulomatous ulcers of the small intestine causing annular strictures and intestinal obstruction in South Indian patients. Resection of the bowel in one of the cases made a histological examination of the ulcer possible. A section of the ulcer showed plasma-cells and lymphocytes, with no evidence of tuberculous granulation tissue.

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CARCINOMA OF THE ŒSOPHAGUS: RESECTION AND ŒSOPHAGO-GASTROSTOMY

By VERNON C. THOMPSON, London

It is now thirty-one years since Torek (1913), first successfully removed a carcinoma of the œsophagus by the transthoracic approach; this patient survived for thirteen years with an œsophageal fistula in the neck connected to a gastrostomy by a rubber tube, and died at the age of 80 from pneumonia (Torek, 1927). Since then many ingenious methods of œsophagectomy have been attempted; a few, such as the pull-through technique first employed by Grey Turner (1933), have been occasionally successful, but the Torek operation with slight modifications remains to-day the standard operation for growths in the middle third of the œsophagus. Even so it cannot be claimed that any great success has been achieved. In thirty years certainly less than 100 operations of this type would seem to have succeeded. Ochsner and De Bakey (1941), in a very extensive review of the literature, state that of 58 cases reported, 41 died. Since 1941, however, several additional cases have been reported.

For growths of the lower third of the œsophagus the ideal procedure is resection of the growth with immediate restoration of continuity by anastomosis of the cut end of the œsophagus to the stomach. In 1908, Voelcker reported a successful resection of the lower end of the œsophagus with œsophago-gastrostomy through an abdominal incision. Ochsner and De Bakey (1941) stated that there had been 24 recoveries reported in the literature from this type of operation.

Resection and œsophago-gastrostomy by the transthoracic route is a more modern development and appears to coincide with the advances that have been made in recent years in thoracic surgery. Successful operations by a combined abdomino-thoracic approach were reported by Oshawa (1933). The first successful resection of carcinoma of the œsophagus with immediate œsophago-gastrostomy by the pure transthoracic route, was reported from America by Adams and Phemister (1938). Since then several cases have been reported in the American literature, and 5 successful operations by the abdomino-thoracic route have been reported by Wu and Loucks (1942) from China.

The patient whose case history is here described was shown at a meeting of the Clinical Section of the Royal Society of Medicine on October 8, 1943 (Thompson, 1943). On the same occasion Tubbs (1943) showed a patient on whom he had performed a similar operation for carcinoma of the cardia involving the lower end of the œsophagus. Steele (1943) has also reported a successful resection with intrathoracic anastomosis for carcinoma of the œsophagus.

CASE REPORT

History.—J. W., a male labourer, aged 54. In June, 1941, while eating fish and chips, he noticed sudden difficulty in swallowing. From that day he continued to have difficulty and was forced to take a soft diet. On four occasions he regurgitated food. He lost 1 st. in weight.

On Sept. 2, 1941, he was admitted to the London Hospital, where a barium swallow revealed an obstruction at the lower end of the œsophagus. He was

Fig. 315.—Radiograph showing irregular stricture at lower end of œsophagus, passing below the diaphragm and involving the stomach.

referred by Dr. Horace Evans to the Department of Thoracic Surgery and transferred to an E.M.S. Thoracic Unit on Sept. 29.

Examination.—A thin but otherwise healthy looking man. All his teeth had been extracted but he wore satisfactory dentures. He was unable to swallow solids, but could manage fluids and semi-solid foods.

The chest appeared emphysematous, but apart from this no abnormality was detected in the heart and lungs. The vital capacity was 2600. The blood-pressure was 115/95. The abdomen was scaphoid in appearance, but otherwise normal. The urine contained no abnormal constituents. The hæmoglobin was 100 per cent. W.B.C., 13,400.

A plain radiograph of the chest showed that the lungs appeared emphysematous; and there was evidence of some pleural involvement at the left base. A barium swallow demonstrated an irregular stricture of the lower end of the œsophagus, passing beyond the diaphragm into the stomach (*Fig.* 315). The radiograph suggested a tumour, the outlines and extent of which were clearly shown to involve the œsophagus and the cardia.

OPERATIONS.—

First Operation.—On Oct. 7, under nitrous oxide, oxygen, and cyclopropane anæsthesia given by Dr. G. Hochschild, an exploratory laparotomy and jejunostomy were performed. A hard mass about 1½ in. in diameter was felt in the cardia extending through the diaphragm. No glandular or hepatic metastases were discovered. A Witzel jejunostomy was performed in the highest mobile part of the jejunum.

The patient was given peptonized milk by the jejunostomy from the first day, to supplement the meals he was able to take by mouth.

Forty-eight hours after operation his temperature rose to 102° and he complained of pain in his left chest. The following day he developed cough and sputum and there was evidence of consolidation in the left lower lobe. Breathing exercises, expectorants, and carbon-dioxide inhalations were given, and by the tenth day his pulmonary symptoms had subsided.

On Oct. 28 an attempt to induce an artificial pneumothorax failed, the lung appearing to be everywhere adherent.

Second Operation.—On Oct. 29, under nitrous oxide, oxygen, and cyclopropane anæsthesia given by Dr. A. I. Parry Brown, the œsophagus was explored by the transthoracic route. With the patient in the right lateral position, the left 9th rib was resected and by means of a mechanical rib spreader the chest was widely opened. The lung was uniformly adherent to the chest wall and diaphragm. The adhesions were separated with ease until an adequate exposure of the mediastinum behind the heart was obtained. The lung was displaced upwards and the mediastinum opened by dividing the pulmonary ligament. A tumour was found involving the lower 1½ in. of the œsophagus and passing below the diaphragm. The healthy œsophagus above the growth was isolated and a piece of tape passed round it. The phrenic nerve was crushed. The growth was then freed, mainly by blunt dissection, but partly by cutting between artery forceps.

The diaphragm was incised for about 4 in. from the periphery to the œsophageal hiatus. The stomach, the spleen, and the left lobe of the liver were found to be adherent to the diaphragm. When these adhesions had been separated the growth was found to involve 1½ in. of the cardia. The left gastric artery was divided, and the upper part of the stomach was brought up into the thorax. The tumour-bearing area was then resected by dividing the stomach and the œsophagus between intestinal clamps. The stomach was closed by a double layer of continuous catgut sutures. An attempt to approximate the fundus of the stomach to the cut end of the œsophagus showed that there would be very considerable tension on the anastomotic sutures. This difficulty was mainly due to adhesions round the spleen, which tended to drag the stomach back into the abdomen. The spleen was therefore removed and this resulted in a much easier approximation.

A separate incision 2 cm. long was then made in the fundus and the cut end of the œsophagus was implanted into the stomach through this incision. Four anchoring mattress sutures of linen thread were applied at equal quadrants, taking all layers of stomach and œsophagus. The incision in the stomach was then closed round the œsophagus, using two layers of seromuscular to muscular catgut sutures. Finally, a piece of omentum was wrapped round the suture line and stitched in position. The incision in the diaphragm was sutured and closed round the stomach.

The chest was closed in the usual manner, an intercostal under-water drain being inserted.

THE SPECIMEN.—The specimen consists of a segment of the œsophagus and stomach measuring 5 in. in length. The proximal ½ in. is composed of healthy œsophagus, the distal 1 in. of healthy gastric wall. The rest of the specimen consists of a hard, ulcerated, craggy growth involving the whole circumference of the œsophagus and stomach and infiltrating the whole thickness of their walls, but not extending beyond the muscular coats. Four small lymph-nodes are attached to the specimen. (*Fig.* 316.)

FIG. 316.—Photograph of the specimen removed.

Histological examination showed the growth to be a squamous carcinoma with considerable keratinization. Of the four lymph-nodes, one contained a little growth.

POST-OPERATIVE COURSE AND MANAGEMENT.—A blood transfusion by the drip method, which was started at the commencement of the operation, was continued when the patient returned to the ward until 1500 c.c. of blood had been given. Three litres of intravenous fluid were given daily until the fifth day, in the form of one litre of 5 per cent glucose in physiological saline and two litres of 5 per cent glucose in distilled water. Feeding by the jejunostomy was started on the second day. Nothing was given by mouth until the third day, when he started to take small quantities of water. The jejunostomy feeds were continued and the amount taken by mouth gradually increased; but no solids were given for two weeks. The jejunostomy tube was removed at the end of a month and thereafter the wound closed at once without leakage. He was then given a normal diet and was able to eat a beefsteak with great relish.

His convalescence after the operation was quite uneventful; drainage from the intercostal tube practically ceased on the sixth day. The underwater seal was discontinued on the tenth day, and the tube was gradually shortened until it was finally removed one month later.

In spite of the fact that he had developed pulmonary complications after the operation of jejunostomy, no similar complications followed the major operation.

An X-ray examination on Dec. 16, 1941, showed the fundus of the stomach above the diaphragm, the stoma appeared satisfactory, and there was no delay at the lower end of the œsophagus (*Fig.* 317).

FIG. 317.—Radiograph showing the condition after operation. The fundus of the stomach is above the diaphragm.

He was discharged from hospital on Jan. 16, 1942.

AFTER-HISTORY.—The patient was subsequently examined at intervals. Soon after he left hospital he developed arthritis in both shoulders, which prevented him from returning to work; but this condition responded slowly to treatment and from September, 1942, to September, 1943, he worked regularly in a factory for 12 hours a day, 6 days a week. He complained occasionally of slight abdominal discomfort after a heavy meal; but he ate anything he chose. On only one occasion did he notice difficulty in swallowing; this occurred in November, 1942, when his gums were sore and he was unable to wear his dentures and chew his food. A barium swallow in May, 1943, showed that there was no delay at the stoma, which appeared satisfactory.

In October, 1943, he developed an attack of acute bronchitis and had to cease work. He recovered from this attack in ten days, but was unable to return to work on account of general weakness.

On Nov. 26, 1943, he was re-admitted to hospital complaining of pain on micturition and defæcation, loss of appetite, and general weakness. His swallowing was normal. On examination he appeared very wasted. There was no obvious involvement of supraclavicular lymph-nodes. The liver could not be felt and there were no signs of free fluid in the abdomen. On rectal examination a hard, fixed, tender mass was palpable in the rectovesical pouch.

On Dec. 16 he died, two years and six days after his operation.

AT AUTOPSY (Dr. W. W. Woods, The London Hospital—P.M. 242/43).—*Bronchopneumonia. Wasting. Secondary carcinoma of stomach, lymphatic glands, and peritoneum.*

Old healed anastomosis between œsophagus and stomach at a point 22 cm. from the lower border of the cricoid cartilage and a few cm. above the diaphragm; very thin scar at the stoma (3·5 cm. in circumference); wall of œsophagus and stomach at the anastomosis flexible, unthickened, and entirely free from growth. No dilatation or hypertrophy of œsophagus above the anastomosis; circumference of œsophagus 3·7 cm. in the lower part and 3 cm. in the upper part; œsophageal muscularis 0·25 cm. thick. Very little secondary growth in the thorax: one lower mediastinal gland (1·0 × 0·7 cm.) full of growth, and a few nodules (0·5 × 0·3 cm.) in one gland near the tracheal bifurcation. Slightly ulcerated plaque of secondary growth (10 × 5·5 cm.) in stomach, saddling lesser curve, its upper end being separated from the anastomosis by a stretch of stomach wall (1 cm. long) free from growth, its lower end being 8 cm. from pyloric sphincter; extension of this gastric growth into lesser omentum as an area of diffuse carcinomatous infiltration, and into pancreas and left suprarenal as small superficial areas. Confluent nodules and plaques of growth in serosa of stomach and in greater part of peritoneal surface of diaphragm, a few (0·3 cm. diam.) in pleura of right dome of diaphragm, a few (1 cm. diam.) scattered in parietal peritoneum and in serosa of large intestine, especially around appendix; thin plaques of growth covered with organizing fibrin in rectovesical pouch. No ascites. Growth throughout a bunch (4 × 2 cm.) of cœliac glands, a few glands (1 cm. diam.) along greater curve of stomach, and numerous lumbar glands (4·5 × 2·2 cm.). About 25 nodules of growth (0·8 cm. diam.) scattered in liver. A few small flecks of pink marrow in shaft and neck of femur. Red marrow in sternum. Obliteration of lower two-thirds of right pleural cavity, and all left, by fibrous adhesions; adhesions very tough over the operation scar in seventh left intercostal space; cutaneous part of this scar a very thin line (18 cm. long). Emphysematous cysts (1 cm. diam.), anthracosis and slight fibrosis in apex of each lung. Calcareous nodule (0·3 cm. diam.) in gland of bifurcation of trachea. Œdema of lungs. Numerous nodules of bronchopneumonia in lower lobe of right lung. Omentum adherent to a left upper paramedian laparotomy scar. Spleen absent. No accessory spleen. Brown atrophy of heart. Very wasted man.

Microscopical sections were made of lumbar glands and liver. They show squamous-cell carcinoma mostly composed of considerably atypical cells, though also containing large areas of keratinization.

DISCUSSION

1. The Incision.—The fact that this patient developed pneumonia after a simple laparotomy and jejunostomy, and yet had no infection of the lung after the major operation by a thoracic approach, strongly supports the belief that pulmonary complications are more likely to occur after abdominal than after chest incisions.

This experience is a strong argument against the abdominal or the combined abdomino-thoracic approach; particularly as the exposure obtained was excellent throughout and no difficulty was encountered on that account.

2. Preliminary Jejunostomy.—Most of the patients with carcinoma of the œsophagus are wasted and are often dehydrated. A preliminary jejunostomy is a great advantage in feeding both before and after the resection of the growth. Moreover, laparotomy allows an adequate examination of the liver and subdiaphragmatic region. A gastrostomy is not desirable as it interferes with the subsequent mobilization of the stomach.

3. The Anastomosis.—Since a paper was published by Carter and his associates (1941) recording their experiences with two patients and their subsequent experimental work on dogs, the method of implanting the œsophagus into the stomach has not been regarded with favour. In their experiments no sutures were passed through the œsophageal mucosa, and the free end of the œsophagus retracted and formed a stricture. If the end of the œsophagus is 'splayed out' inside the stomach by through-and-through mattress sutures, as in the operation here described, retraction is prevented. In this case in spite of removing the spleen there was some tension on the anastomosis and it is probable that if the implantation method had not been employed the anastomosis would have leaked.

At post-mortem examination the stoma was 3·5 cm. in circumference; it was represented by a thin pliable scar with no undue fibrosis or induration. Regurgitation of gastric contents after œsophagogastrostomy, particularly when the patient is lying down, has been reported by Phemister (1942) and others. These symptoms were not experienced at any time by the writer's patient. It would seem that regurgitation is most likely to occur through a large stoma and that a narrow stoma is an advantage, providing it causes no delay to the passage of food.

4. Scope of the Operation.—The operation described has proved to be a practical and successful procedure. Its frequency and success in the future depend, like operations for carcinoma in other parts of the body, on early diagnosis.

The Registrar-General (1944) gives the following statistics for deaths from cancer in England and Wales for the years, 1920, 1930, and 1940.

Year	*All sites*	*Œsophagus*	*Ratio of Œsophagus to All Sites*
1920	42,420	1,705	0·040
1930	56,204	2,217	0·039
1940	68,740	2,321	0·034

These figures show that although the number of deaths from carcinoma of the œsophagus is increasing, the ratio of these deaths to deaths from carcinoma of all sites has diminished since 1920.

Ochsner and De Bakey (1941), basing their figures on a collected series of 8572 cases, state that 42·8 per cent of carcinomas of the œsophagus occur in the lower third. It would thus appear that in England and Wales in 1940 there were about 1000 persons who might have been given a chance of survival by the type of operation described.

Carcinoma of the cardiac end of the stomach also presents a field in which the same operation can be applied.

SUMMARY

A case of carcinoma of the lower end of the œsophagus involving the stomach is described. After preliminary jejunostomy, the growth was resected and œsophago-gastrostomy was performed through a thoracic incision. The patient swallowed normally until his death 2 years and 6 weeks later from multiple intraperitoneal metastases.

The operation was performed in October, 1941, and would appear to be the first successful one of its kind carried out in this country.

I wish to express my thanks and appreciation of their help to Dr. G. Hochschild and Dr. A. I. Parry Brown for the anæsthetics; to Dr. L. C. Blair for the X rays; to Dr. W. W. Woods for the post-mortem report; and to my house surgeon, Mr. H. G. Eastcott, for his careful attention to detail in the pre- and post-operative treatment of the patient.

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PRELIMINARY REPORT ON THE USE OF WHOLE SKIN-GRAFTS AS A SUBSTITUTE FOR FASCIAL SUTURES IN THE TREATMENT OF HERNIÆ

By GEORGE B. MAIR

WOODEND AND OLDMILL E.M.S. HOSPITALS, ABERDEEN

Brigadier Edwards (1943), in this Journal, has exposed in masterly fashion the modern views on the treatment of inguinal herniæ.

The position is that the Bassini operation, with all its modifications, is not only inefficient, but actually harmful, and should be abandoned. In its stead there are two alternatives: simple herniotomy, where high excision of the sac is combined with repair of the torn or weakened fascia transversalis; and the second, or plastic, where living fascial sutures are used to narrow the internal abdominal ring, and to strengthen the weakened posterior wall of the inguinal triangle.

Edwards's paper should be read as a background to this, which is distinctly a preliminary report suggesting that the use of whole skin-grafts inlaid under extreme tension and in such a fashion as to protect the posterior wall of the inguinal canal, and narrow the internal ring, may be superior to fascia in most respects. The method has been used for cases where fascia was formerly indicated, and a history of the investigations which have been in progress in Aberdeen during the past year will be detailed.

First, however, it is necessary to discuss the position which the use of fascia holds, and to note its advantages and disadvantages.

Use of Fascia in Herniæ.—The indications for the use of fascial sutures are:—

1. All recurrent herniæ of any type.
2. All primary direct inguinal herniæ.
3. All primary oblique inguinal herniæ where there is muscle atrophy and weakness of rings and posterior wall.
4. All funicular or 'saddle bag' types.
5. As the final stage in the Mayo repair of umbilical herniæ.
6. As the final step in the repair of large ventral or incisional herniæ.

It is thus recognized that fascia is reserved for those cases which are most likely to recur, and in those subjects whose physique is prejudicial to the success of a physiological repair. It might then be expected that cases dealt with by fascia would show the higher recurrence rate.

Follow-up recurrence figures show wide variation in different clinics and are influenced by many variable factors. Despite the difficulties attendant upon acceptance of many published figures, certain facts emerge clear. The recurrence rate varies in the case of indirect inguinal hernia up to 30 per cent, with an accepted average of 12 per cent for the Bassini operation.

McCloskey and Lehman (1940) analysed the results in different American clinics, and found that with fascia, recurrences varied in different clinics from between 3 and 4 per cent for the indirect, to between 7 and 9 per cent for the direct, varieties. These figures suggest that fascia, though it be used in the less favourable type of case, definitely reduces the incidence of recurrence.

Much depends upon the time interval before re-examination. It is accepted that at least 75 per cent recur within the first year, and at least 60 per cent within the first six months (Page, 1943; Edwards, 1943; Judd, 1908; Erdman, 1923).

Fascia does, however, have certain disadvantages:—

1. The McArthur method does not give enough fascia to fulfil the requirements of the average case, and is applicable mainly to the inguinal type. For a large hernia the method is not ideal.

2. The Gallie technique involves either an extensive wound in the thigh, or a small incision combined with the use of a fasciotome. In either case there is a risk of post-operative pain referred to the thigh and hip-joint of the affected side, and the possibility of obvious muscle hernia. This pain may be quite intractable, is aggravated by exercise, and, in my experience, occurs in 25 per cent of subjects. The aetiology of this pain is obscure, and in those cases where a workman's compensation suit is impending, or in Service personnel, it may be a great annoyance.

3. Infection of the wound in the abdomen is fairly common. Bendick (1937) found sepsis to be present in 7·9 per cent of his cases where autogenous fascia had been used, and in 12·1 per cent where ox fascia was used.

4. Fascia will unite to fascia under considerable tension (Law, 1924) and it may be applied under tension to unite the conjoined tendon to Poupart's ligament, or, it may be darned loosely between the two structures. In either case there are potential gaps between the strands of fascia, and direct recurrences may develop.

In several cases operated on by myself, the sac had clearly insinuated its way through a gap in the fascial strands which in other parts had effected a powerful reinforcement.

5. The needle used for the Gallie method is large, and apt to traumatize the inguinal ligament. There is also a risk of penetrating the femoral vein. This complication has been frequently

mentioned in the literature (Robitshek, 1925), and in one example which came to my notice death resulted on the operating table.

6. Finally, there remains a substantial recurrence rate. Even the best figures available are too high, and provide room for consideration as to how they may further be reduced.

Cutis Graft Repair.—Rehn (1914) and Loewe (1913) advocated the 'cutis graft method for the repair of anatomical defects in post-operative herniæ,' and, of these, Rehn was the first to apply the principle clinically. Cutis contains all the elements of the skin excepting the epidermis, it is elastic, inherently active, and composed of a rich network of connective-tissue fibres. After transplantation, according to Uihlein (1939), these inherent factors persist in the metamorphosis of the graft, and, owing to the stimulus initiated and maintained by the tension under which the graft is sutured, a metaplasia into stout connective tissue takes place.

Rehn and Schwartz, quoted by Uihlein (1939), performed a tenotomy of the tendo Achillis in a dog, and repaired the defect with cutis graft. They found that a gradual metamorphosis took place after ten weeks into a tendon difficult to differentiate from ordinary tendon.

Rehn applied his cutis graft, which he took from the thigh, into the defect requiring plastic repair, and attached it under a maximum degree of tension to its surrounding structures by interrupted sutures. The graft was cleared so far as possible of epidermis.

Uihlein operated upon two patients who had submitted to a cutis graft repair four years earlier. Sections removed for examination showed a complete metamorphosis of the transplant. The grafts represented normal connective tissue with its fibrous and fatty components, blood-vessels, and nerves. No hair follicles or glandular tissue could be identified, and there was no evidence of cyst formation, either macroscopical or microscopical. No scar tissue was seen in the sections examined.

Rehn used this method in 65 gross post-operative herniæ with 6 poor late results. There were a further 39 operations where cutis was used to fill in a tissue defect, and of the total of 104 operations for various purposes, 15 were complicated by wound sepsis.

Cannaday (1943) and his surgical associates in the Charleston General Hospital used the method in 37 cases, of which 27 were herniæ of various types. These workers applaud the principle, state that cutis is superior to fascia, find that the graft heals firmly and promptly, and commend the fact that it is easily obtainable in portions as large as necessary for any given case.

Neither Cannaday and his co-workers nor Rehn and his associates have investigated with detail the histology of their grafts, but they are agreed that clinically the results are excellent. Certain plastic surgeons (Eitner, 1920; Peer and Paddock, 1937; Peer, 1940), however, have studied the subject at length, but only in buried implants not sutured under tension. They found that despite every attempt to remove the epidermis, apices of hair follicles and sweat glands remained in the grafts, and that when implanted, these structures disappeared and the grafts were gradually transformed into fibrous tissue. The grafts fused with their surroundings through the mechanism of inflammation and repair associated with aseptic wound healing.

The investigations raised the old question as to the danger of epidermoid cyst formation, and this is an objection which is frequently raised to the use of cutis implants.

There is insufficient space in this paper to discuss this problem, but work done suggests that the risk of an inclusion cyst is negligible when the graft has been inlaid under tension (Rehn, 1914; Loewe, 1913; Uihlein, 1939; Cannaday, 1943; Peer and Paddock, 1937). In buried epidermal strips, however, merely inlaid into muscle or fat, and not under tension, inclusion cysts may be found (Garré, 1894; Kaufman, 1884; Schweninger, 1884; Hesse, 1912; Pels-Leusden, 1905; Zimches, 1931).

The method adopted by Rehn has clinically one decided disadvantage. The prepared thigh has an area of epidermis removed from it by a Thiersch razor, and from this denuded part the cutis graft is cut. Edges of the resulting wound are devoid of epidermis, and are closed as well as possible. Yet healing is slow, and, in any case, time is lost in the preparation of the graft.

The question arose in the author's mind as to whether it was really necessary to remove the epidermis, and with that end in view he investigated the use of whole-skin implants sutured under tension, in rabbits and in guinea-pigs. At the same time the operation has been applied clinically to a considerable number of herniæ where fascia otherwise would have been used. The results to date will be shortly indicated.

WHOLE SKIN-GRAFT IMPLANTS

In 1938 the author, whilst repairing a very large ventral hernia in a woman aged 64, for the first time implanted a large whole skin-graft, having excised and closed the redundant peritoneum. The graft was sutured to the anterior aspects of both rectus sheaths and to the linea alba. The graft measured some 4 in. by 5 in., and was taken from redundant skin excised by an elliptical skin incision. The repair was firm and satisfactory. Eighteen months later there was no recurrence or other complication. Between 1938 and October, 1943, a similar method of repair was adopted in 7 large ventral, 3 umbilical, and 1 epigastric hernia. In none was there any complication during the early post-operative period, but unfortunately, owing to the war, none of the cases could be followed up

for more than a few months. The immediate results, however, had been so encouraging that it was determined to apply the same method to the repair of inguinal herniæ and to investigate the histology of the implants.

The rabbit was chosen as being a suitable animal for the work. An incision was made over the lumbar vertebral spines, the lumbodorsal fascia exposed and a portion excised. The defect was repaired by a whole skin-graft from which hair had been shaved, cut from the edges of the wound, and sutured under a high degree of tension to the adjacent aponeurosis. Sections of normal rabbit skin were studied for comparative purposes.

Five months later, it was found that sebaceous glands, hair follicles, and epidermis had entirely disappeared, that hairs had been surrounded by a massive foreign-body reaction, and that this in turn was being encapsulated off by a firm fibrous stroma. Macroscopically at five months it was very difficult to determine the margin of the graft, this being so firmly attached to its surroundings and so closely resembling dense normal fascia in appearance. The degree of metaplasia was striking.

Microscopically, however, dermal elements could be identified apart from the hair remnants. Atrophied sweat glands were detected, and a few dermal papillæ in a similar state of degeneration. The dermis showed a dense infiltration with fibrous tissue and impressive degree of vascularity. The margins of the graft were firmly united to adjacent fibrous tissue, and vascular septæ infiltrated deeply from the graft to subjacent muscle. A number of small, rounded, and encapsulated structures were noted which appeared to be masses of cornified epithelium arranged in whorls.

In sections studied after three weeks, the graft had materially altered macroscopically and closely resembled white fibrous tissue. Microscopically all elements of the skin could be identified, but a fibroblastic reaction was in process and granulation tissue apparent at the margins of the implant. No trace of epidermoid cyst could be detected.

Sections removed at other stages showed features intermittent between the two extremes described.

The opportunity arose to explore an inguinal canal in the human subject into which a whole skin-graft had been sutured for a large direct hernia three months earlier. The graft was very firmly attached to Poupart's ligament, aponeurotic medial aspect of the internal oblique, and insertion of rectus sheath in the pubis. It was not possible to identify with assurance where the graft ended and normal tissues began as the degree of metaplasia into tissue closely resembling that to which the implant was sutured was so striking. The appearance closely approached that of a normal inguinal canal, and the dense fibrous-tissue formation and deformity of normal anatomy, which is so striking a feature of recurrent fascial herniæ, was absent. A section was removed for examination. The histology was that of highly vascular connective tissue, in which no dermal or epidermal elements could be identified. The graft was richly infiltrated with collagen and elastic fibres, and there was firm adhesion to the underlying muscle. No trace of even microscopic cyst formation was found, but a number of giant cells were observed. No hair remnants could be identified.

The animal experiments are being continued, and in a later report the findings will be submitted. No contra-indication to the use of the grafts in the human subject has been found by this method to date.

CLINICAL APPLICATION OF THE PRINCIPLE

In a series of 70 inguinal herniæ, direct, indirect, and recurrent, 6 umbilical, 10 ventral, 1 epigastric, and 1 femoral, repair has been performed by means of a whole skin-graft. There have been 2 cases of mild sepsis which have cleared up, the first under three weeks and the second under four. One scrotal hæmatoma developed and there have been 2 cases of

Table I.—Operations in which Whole Skin-grafts were Used

Type of Hernia	Number	Average Age
Indirect inguinal	45	43·4
Recurrent indirect inguinal	2	27·5
Direct inguinal	16	41·2
Indirect-directing	7	46·5
Umbilical	5	44·0
Recurrent umbilical	1	55·0
Ventral	10	39·9
Epigastric	1	38·0
Femoral	1	39·0

Table II.—Length of Time since Operation, up to July 31st, 1944

Months	1	2	3	4	5	6	7	8	9	12	15	18	24	24–36	36–48	60
Number	8	4	13	7	13	9	5	10	6	1	1	1	2	3	4	1

Table III.—Complications

Sepsis	2 cases. Cleared, 21–28 days
Chests	2 cases bronchitis, mild
Others	One scrotal hæmatoma. Cleared

post-operative bronchitis, which cleared up within a few days.

The results have been satisfactory, but, of course, much more time must elapse before recurrence figures can be submitted.

TECHNIQUE OF OPERATION

Skin Preparation.—It is suggested that the skin chosen for the purpose, coming as it does from the vicinity of the symphysis pubis, is potentially infected, and that even if the surface be sterile, organisms may lurk in the depths of the hair follicles. Proof of the safety of using this skin lies in the results of experience. The method has been used in 88 cases with impunity.

The method of skin preparation is first of all by careful shaving, followed by scrubbing with ether soap and warm water for ten minutes. Spirit compresses and a sterile double hip spica bandage are then applied. The procedure is repeated daily for three days in all.

Before operation, in most of my cases, a culture of organisms from the skin was taken. Only a few showed any growth, and these mainly scanty colonies of *Staph. albus*.

Anæsthetic.—Gas, oxygen, and ether, with preliminary premedication of omnopon and scopolamine 3–4 c.c. is entirely satisfactory. For ideal relaxation an inhalation anæsthetic is essential.

Incision.—The incision is fashioned to enclose an ellipse of skin which is at least 2 in. long and approximately 1 in. at the broadest diamater. It extends from the symphysis pubis upwards and laterally, parallel to Poupart's ligament, for 3 or 4 in.

The skin with attached subcutaneous tissue is removed and immersed in warm normal saline solution until required.

The external oblique is incised in the usual way and the flaps reflected to expose the inguinal canal and spermatic cord. The superior flap of the aponeurosis is detached for a distance of approximately ½ in. from its pubic connexion, in order the better to expose the area to which the graft will be sutured medially. This having been done, the cord with cremaster muscle is lifted from the canal, and both are separated clearly from the floor, and from adhesion to the inguinal ligament, from the internal ring to the pubic end. It is essential to clear the area thoroughly, and this is readily done by clean firm sweeps with a gauze swab (Pitzman, 1919).

The cremaster and coverings of the cord are incised in the usual way, the sac dissected clear, and removed by ligation and excision at as high a level as possible.

The next step is to buttress the fascia transversalis, and to narrow the pillars of the internal ring by carefully inserted sutures, thus forming a firm support for the graft.

The repair of the incision in the coverings of the cord is made with a running suture, and attention turned to the graft.

The ellipse of skin will be found to have shrunk considerably in size. The extremities are seized by Allis forceps and the subcutaneous fat rapidly dissected away. The pointed extremities of the ellipse are then chopped off, so as to leave a free end of at least ⅓ in. at one end, and ¼ in. at the other. The larger end is then incised vertically along from its midpoint for a distance of approximately ½ in. to ¾ in., and each tail of skin grasped at its tip by Allis forceps. The whole is then laid into the inguinal canal and the inferior forceps placed below the retracted cord. The forceps are then pulled upwards and laterally so that the apex of the V thus formed grips the internal ring.

The medial margin of the graft is sutured by three stitches—to the lower anterior aspect of the rectus sheath, the fascia over the symphysis pubis, and the extreme medial edge of Poupart's ligament. Care is taken that the graft is stretched taut and that it well overlaps the pubis and rectus.

The superior Allis forceps are then drawn gently but firmly upwards and laterally to stretch the superior edge of the graft tightly. The end of the skin pedicle is now sutured to the medial aponeurotic expansion of the internal oblique. The lower skin pedicle is then also drawn outwards and the extremity sutured to the shelving inner margin of Poupart's ligament at least ½ in. above the level of the internal ring. Sutures are placed at intervals of about ¼ in. along the lower edge of the graft to anchor it to Poupart's ligament, and care is taken that the ligament is sutured as near the floor of the canal as possible. The final row is applied to the superior edge of the graft, and fixes it to the aponeurotic medial edge of the internal oblique very close to the lateral margin of the rectus sheath.

The outer aspect of the graft is then sutured, joining the two pedicles together lateral to the internal ring, and in such a fashion as to form a skin plaque lateral to it as well as medial, forming another ring of skin over the internal muscular ring, and causing the cord to arch slightly upwards in its course from the ring to the canal. (*Fig.* 318.)

In inserting the superior row of sutures and those joining the two skin pedicles, care is taken to ensure that the graft is sutured under great tension to its attachments. Thirty-day chromicized catgut is used for suturing the graft.

A fine layer of sulphonamide powder is insufflated on to the surface of the graft and the surrounding tissues, the external oblique is closed, and the skin sutured with interrupted fine silk gut.

The patient is allowed up on the twentieth day. It is established that fascia requires three weeks to heal firmly, and there can be no point in permitting freedom of movement before that day.

In this description of technique no mention has been made of the many difficulties which may confront the operator. But it is emphasized

that as the whole skin-graft is advocated for use in large and difficult cases, it follows that difficulties will be encountered, and they are all readily capable of being surmounted by this new method.

The same principles can be adopted for direct herniæ, and can easily be used as a final reinforcement for umbilical, ventral, or epigastric herniæ,

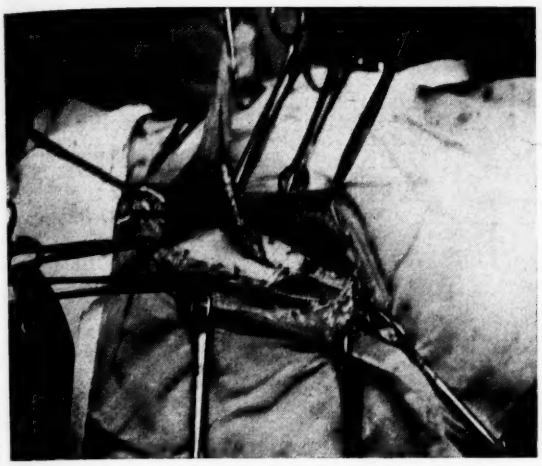

FIG. 318.—The graft has been sutured into position. The flaps of the external oblique aponeurosis are held retracted by mosquito forceps. Note the two skin pedicles stitched together lateral to the internal ring, and that the graft is stretched tightly over the entire posterior wall of the canal.

and in fact, one of the main attractions is the general applicability of this method to any type of hernia.

COMMENT ON THE OPERATION

It must be recognized that a certain amount of experience with the method is necessary before mastering the technique. So far as duration of operation is concerned, in my first seven or eight cases my time was generally fifty minutes. Since then it has become less, and for the last fifty I have rarely required over half an hour to perform any variety of primary inguinal herniorrhaphy. Recurrent herniæ, of course, take somewhat longer.

When correctly bestowed, the graft affords protection to the entire inguinal canal, and narrows the internal ring, but care must be taken not to narrow it too much lest later varicocele or testicular atrophy develops. The apex of the V in the graft should fit snugly into the muscle of the internal ring on its inferior aspect, and the cord should emerge smoothly, but with a slightly altered direction, from the ring over the edge of the skin-graft, into the canal.

In preparing the bed for the graft it is essential to obtain a thoroughly good view of the anterior aspect of the symphysis and the lower margin of the rectus abdominis. If attention is given to these points, the graft will give protection to the medial margin of the canal where direct hernia is so prone to develop.

In my experience the operation takes less time to perform than a fascial repair, and the finished immediate result appears to be stronger than that presented by fascia.

There is never any difficulty in obtaining closure of the skin wound even after removal of the graft.

Finally, my impressions to date are that the method is excellent, and that post-operative complications are fewer than when fascia is used. It is too early to give figures of recurrence or late post-operative morbidity, and it is again emphasized that this is a preliminary report. Detailed analysis of the results will be presented in due course, and compared with those for fascial repairs, Bassini operations, and simple herniotomies, as performed in Woodend and Oldmill hospitals.

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COMPLETE RUPTURE OF THE PANCREAS

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ANY type of injury of the pancreas is rare, and recently I have had the opportunity of operating on a young man with a complete rupture of the pancreas due to a subcutaneous injury, an excellent recovery taking place.

CASE REPORT

HISTORY.—J. C. W., a male aged 20, of slim build but good physique, was admitted to Leith Hospital on Dec. 21, 1943. In the course of his duties he was walking backwards in the rear of a reversing lorry, directing the driver, when he was unexpectedly pinned between the back of this lorry and the back of a stationary lorry. The lower part of the chest and the upper abdomen were forcibly compressed, but only for a matter of seconds, as the driver at once noticed the danger and released the pressure by moving forwards. A momentary severe pain was felt at the time of the impact, but it passed off immediately. The patient was able to walk away from the scene of the accident, was then driven to the hospital nearby, and actually walked unaided through its doors half an hour after the injury.

ON EXAMINATION.—There was no sign of any vertebral, rib, or other bony injury, and there was no external bruising of the chest or abdomen. A moderate degree of shock was present, the pulse-rate was 80 per minute, the temperature 97·4°, and the respirations were 30 per minute, shallow, and painful if encouraged to be deep. There was a complaint of epigastric discomfort and of slight pain in the right shoulder. The movements of the abdominal wall were restricted during respiration, tenderness was present in the epigastrium, and there was slight generalized guarding of all the abdominal muscles. The urine was normal.

The patient was at once admitted to a surgical ward and the state of shock passed off without any special methods of treatment. A careful watch was kept and the condition remained quite satisfactory for three hours. Thereafter, the pulse-rate rose in the next hour from 80 to 90, and the following hour still more rapidly to 110 per minute, during which time a blood transfusion had been started and arrangements made for operation.

Vomiting had never occurred, but the upper abdominal and right shoulder pains were now quite severe. Movements of the abdominal wall during respiration were absent, rigidity and tenderness were generalized, signs of free fluid were demonstrable, and there was no loss of liver dullness. There was thus definite evidence of some severe abdominal injury, but an exact diagnosis could not be made, although a rupture of the liver, spleen, or mesentery was considered most likely. In spite of a continuous blood transfusion, the pulse-rate was still rising, and had reached 120 per minute by the time the operation was started, six hours after the injury.

AT OPERATION.—An open ether anæsthetic was administered, and the abdomen was opened through an upper left paramedian incision. On entering the peritoneal cavity, a tremendous amount of fresh blood was found lying free in all the peritoneal compartments, and, in spite of suction and mopping, the blood rapidly welled up into the wound from every direction, so that at first there was great difficulty in carrying out an exploration. There was no sign of any free gastric or intestinal contents and examination of the liver, spleen, kidneys, and small intestine was negative. A tear 1 in. long was found in the lesser omentum near the stomach, but the stomach was not damaged; bleeding vessels were ligated and the tear was repaired. The inferior surface of the transverse mesocolon was inspected and revealed an irregular rent, about 3 in. long, involving the base of the mesocolon but not reaching the bowel, which was intact. The rent was immediately to the left of the undamaged middle colic artery, but a branch of it was completely severed, calling for ligation of both ends.

Further examination at the base of the mesocolon showed an extraordinary injury. The pancreas was found to be completely split into two parts, as though by a sharp knife, the line of division running vertically almost exactly through the centre of the organ. The two parts had retracted with a gap of fully two inches between them and the left half of the pancreas was lying entirely inferior to the base of the mesocolon and to the left of the rent in it. On the other hand, the right half of the pancreas was superior to the base of the mesocolon and difficult to examine through the rent, and, to expose it better, an incision was made in the greater omentum just distal to the stomach. By using these different methods of access, the two raw ends of the pancreas were approached and a few individual bleeding points were ligated. In view of the wide separation, there seemed little possibility of carrying out a successful end-to-end union, and it did not seem justifiable to prolong the operation by carrying out the alternative procedure of excision of the left half. The simple method was therefore adopted of inserting several deep mattress sutures into each severed end, which had the satisfactory effects of controlling hæmorrhage from the gland and of closing off most of the raw surfaces.

Even yet the total extent of the injury was not discovered, and the hæmorrhage continued, though now much more slowly. Using the approach through the greater omentum, a large vessel, posterior to the pancreas, was found completely severed, with one end lying quite free from its surroundings. This proved to be the splenic vein and its two ends were ligated. There was no damage to the splenic artery, aorta, or any other large vessel.

Five grammes of sterile sulphanilamide powder were applied at the site of injury. A large gauze pack was inserted through the opening in the greater omentum and was firmly placed between the ends of the pancreas, in order to control the slight oozing of blood which persisted and also to act as a drain for the anticipated collection of pancreatic secretion. The rent in the mesocolon was closed as far as possible, and the abdominal incision was firmly closed around the protruding end of the pack.

POST-OPERATIVE COURSE.—The patient stood the operation remarkably well, but nevertheless anxiety was felt on account of the possible formidable complications. The blood transfusion, started before operation and kept up during it, was continued

thereafter. A total of six pints of stored blood was thus administered during twelve hours and it was followed by two pints of plasma. The next day the patient was cheerful and in good condition. On the fourth day the gauze pack was removed, sodden with blood and pancreatic secretion, but no secondary hæmorrhage occurred. It was feared that complications might arise as a result of interference with the blood-supply to the transverse colon, but fortunately this proved unfounded.

SUBSEQUENT PROGRESS.—As was anticipated, a pancreatic fistula developed as a small opening at the site of drainage in the middle of the operation incision. The rest of the wound healed without difficulty and there was a complete absence of any sign of fat necrosis or wound digestion. During the next six weeks there were periodic bouts of upper abdominal pain, and there were several rises in temperature to 100° or over, lasting intermittently for a few days, settling down thereafter, but only to recur later. X-ray examination showed no sign of subphrenic abscess, pulmonary complication, or bony injury.

The type and quantity of the fluid passing from the fistula varied greatly, even from day to day. At times it was profuse, clear, and watery, and contained trypsin but none of the other pancreatic ferments and no bile-pigment. At other times it was thick and definitely purulent, and bacteriological examination showed a heavy infection with *Staph. aureus*, proteus, and *B. coli*. The fistula gradually became smaller and closed finally nine weeks after the operation. Interesting physiological observations on external pancreatic fistula were recently made by Comfort and Priestley (1943), but it was not possible to repeat them accurately on account of the very variable nature of the fluid and its heavy infection. It was noted, however, that the amount of discharge became definitely less on a low protein diet and increased again on an ordinary diet; it was not affected by a fatty or carbohydrate diet. It was not desirable to continue the low protein diet for too long, and it was tried experimentally on a few occasions for periods of three or four days.

At no time was there any obvious evidence of deficiency of the internal pancreatic secretion and glycosuria was never present. It was not considered necessary to carry out a blood-sugar curve. The fæces were examined at different times to investigate any deficiency of the external secretions. Two and a half weeks after the operation, the total fat in the dried fæces was 35 per cent, and four weeks after healing of the fistula it was 16 per cent, showing a possible mild degree of insufficiency at first. On both occasions the proportion of split fats to unsplit fats was 65 per cent to 35 per cent, indicating no loss in satisfactory fat digestion. The general condition remained quite good throughout and the appetite was seldom upset. In the nine weeks when the fistula was present, loss in weight was considerable, there being a fall to 7½ st. from a normal of over 10 st. On closure of the fistula, the weight became restored to normal in the next six weeks.

The only final disability was a small incisional hernia, for which a suitable belt was prescribed.

DISCUSSION

Varieties of Injury.—An injury to the pancreas may naturally be produced by any variety of penetrating or perforating wound of the abdomen, for example by bullets, shrapnel, bayonet, or knife. This type is almost always associated with severe injuries to other viscera and by its nature is often rapidly fatal. It forms a very small proportion of abdominal injuries treated by operation. The pancreas may rarely be damaged at surgical operations, particularly gastrectomy, nephrectomy, or splenectomy, and the diagnosis is to be suspected in the event of untoward complications after a particularly difficult technical procedure.

The subcutaneous type of injury is the most common and is as much a hazard of peace as of war. It is generally caused by some severe compression of the upper abdomen and the common mechanisms include crushing between two vehicles or beneath fallen masonry, running over by the wheel of a vehicle, and a kick or a blow on the unguarded abdominal wall. It is generally believed that the effect of any of these mechanisms is to push the pancreas violently against the rigid vertebral column, although Venable (1932) questioned this idea and put forward the theory of *contre-coup*, the pancreas being unable to transmit the wave force created by a blow through its substance. Various degrees of injury to the pancreas may be produced, such as simple bruising, a partial tear, or a complete tear, and it is possible for spontaneous resolution to follow the mildest traumata. In the rather more severe examples, several possible changes may take place, including acute pancreatitis with severe hæmorrhage or fat necrosis, a generalized peritoneal hæmorrhage, a localized hæmatoma in the lesser sac, acute peritonitis, or a pseudocyst, causing a swelling slowly increasing in size weeks or months later. Further reference to such injuries is omitted in this article.

The most severe injury is a complete division of the pancreas through the middle, the pancreatic duct escaping only in very rare instances. The pancreatic injury tends to be associated with and overshadowed by damage to other structures such as stomach, duodenum, duodeno-jejunal flexure, jejunum, liver, kidney, spleen, transverse mesocolon, and large blood-vessels, especially the splenic vein. These multiple injuries must necessarily be very severe, and it is highly probable that many of the cases are never diagnosed but die almost at once or within a few hours. In those patients who survive the immediate injury, there are no typical signs suggestive of pancreatic trauma, and the manifestations are those of any serious abdominal injury associated with severe hæmorrhage. The estimation of the urinary diastase is probably the only useful laboratory test, a high reading being indicative of a pancreatic lesion.

Previous Examples of Complete Rupture.—The literature on the subject of pancreatic trauma reveals very few instances of total severance of the organ, although there are more references to the less complete cases. What is generally believed to be the first recorded example was that of Travers in 1827, quoted by Moynihan (1926) among others: Following a

run-over accident, autopsy demonstrated a pancreas torn completely across, in association with ruptures of the duodenum, jejunum, and transverse mesocolon, and with many fractured ribs. Mayo Robson and Cammidge (1907) referred to the fatal case of Wilks and Moxon, the pancreas being divided into two parts, and they also reproduced the illustration of a similar specimen in St. Bartholomew's Hospital Museum. Cases of total division of the pancreas, with successful results after operation, were those of Garré in 1905 (quoted by Moynihan), Feist in 1926 (quoted by Bailey, 1944), Newton (1929), and Brown and Barlow (1932), who also quoted the case of Heineke in 1907. It is not advisable to attempt any accurate enumeration of such cases, as several references have not been traced and other references do not indicate clearly the exact extent of the injury.

Treatment.—The correct treatment in all pancreatic injuries is undoubtedly operation, and figures as long ago as those of Mikulicz (1903) show the advantages over non-intervention. In complete division of the pancreas, owing to the severe hæmorrhage present, operation must necessarily be performed early, and Garré, Brown and Barlow, and I myself operated about six hours after the injury and Newton a little over twenty-four hours after. The actual management of the tear in the pancreas varies according to the operator's own views and to the local findings, and success has followed different methods. Drainage either by a gauze pack or by a rubber tube seems essential, either along with methods of suture or possibly as the only method in desperate cases or those with much friability of the pancreas. Newton was able to carry out a direct end-to-end suture of the two portions of the pancreas, the two ends of the duct being merely approximated, not united, and an omental flap being used to encircle the suture line. Garré and Brown and Barlow also performed direct sutures. Walton (1930) had previously indicated that such a method was bound to fail, and suggested the complete removal of the separated body and tail and careful suture of the divided stump of the neck. In my own case, the two ends of the pancreas were so widely separated that end-to-end union was impossible, and the easiest method was therefore to close the two raw ends separately by sutures. After direct end-to-end suture, it seems doubtful if the left half of the pancreas can ever recover its full external secretory functions, as the chance of the duct regaining its normal patency seems remote. However, there does not appear to be an obvious loss of any of the pancreatic functions after the various operations.

The external pancreatic fistula, which is an almost constant sequel, usually closes spontaneously after several weeks or months, but during that time there is a considerable loss in weight. It may give rise to very painful digestion of the wound, and Fast (1930) used a method recommended by Potter (1927) for duodenal fistula, which, it is maintained, allows rapid relief from this complication and early closure of the fistula. A catheter is held in the fistula and packed around with gauze soaked in 10 per cent solution of Witte's peptone; through the catheter hydrochloric acid $N/10$ is instilled into the fistula at the rate of 20 minims per hour. A high caloric diet is given and it is claimed that digestion of the wound ceases in twenty-four hours, although the fistula may not heal for thirty days. In my own case, with an absence of wound digestion and the presence of an intermittent purulent discharge, there was no indication for using this method.

SUMMARY

A report has been given of a young adult male who received a momentary severe crushing of the upper abdomen. At operation, six hours after the accident, a gross degree of intraperitoneal hæmorrhage was present, and there existed a complete division of the pancreas into two parts, with wide separation. The two raw ends were separately closed by sutures, and a large gauze pack was inserted for drainage and hæmostasis. Other injuries were a transection of the splenic vein and tears of the transverse mesocolon and the lesser omentum. An external pancreatic fistula was a sequel, closing spontaneously in nine weeks, and the final result was excellent.

Mention has also been made of 8 previously reported cases of complete rupture of the pancreas, with successful results in 5 cases after operation. Methods of management of the pancreatic injury have been discussed.

I wish to express my thanks to Mr. A. Philp Mitchell, Surgeon, Leith Hospital, for the opportunity of operating on this most interesting abdominal injury, and for much guidance and encouragement in writing this article.

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ISCHÆMIC DAMAGE IN THE PERIPHERAL STUMP OF A DIVIDED NERVE

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IT has been shown by Holmes, Highet, and Seddon (1944) that the nerve lesions found in Volkmann's ischæmic paralysis, apart from those directly due to the injury that produced the vascular damage, are the result of an ischæmia of the nerves, which is most severe where they traverse the zone in which the ischæmia of muscle is maximal. The damaged nerve remains, of course, in continuity, but a gross interference with its normal blood-supply may cause irreparable intraneural change, in the form of an obliterative collagenization within the nerve-bundles themselves.

In the case about to be described the ischæmic damage was confined to the peripheral stump of a divided nerve, the neighbouring muscles being apparently unaffected. This finding was an accidental one, but its significance may well be considerable; the case has, therefore, an interest not determined solely by its rarity.

CASE REPORT

HISTORY.—On March 9, 1942, a Royal Air Force pilot (born in 1916) crashed over enemy-occupied territory and suffered a compound fracture of the upper third of the left radius and ulna, with damage to the median and ulnar nerves. He seems to have been fairly well treated, though the first plaster was too tight and produced two deep sores at the wrist. After 4–5 weeks there was some return of sensation and movement in the fourth and fifth digits.

November, 1942: Wound healed.

July, 1943: Excision of head of radius, with incidental damage to the posterior interosseous and superficial radial nerves. The operation was done with a view to restoring pronation and supination.

Oct. 26: Repatriated.

Dec. 16: Sent by the Royal Air Force to the Wingfield-Morris Hospital for consultation.

ON EXAMINATION.—The wound in the upper third of the forearm lay anterolaterally and there was much deep scarring. The head of the radius had been excised, and the fracture of the upper third of its shaft had united. The ulnar fracture was ununited and there was severe lateral bowing with anterior displacement of the distal fragment.

Median nerve: Paralysis was complete except for a flicker (? trick movement) in flexor pollicis longus and in one or other of the long flexors of the middle finger; there was a typical zone of sensory loss and anhidrosis.

Ulnar nerve: Flexor carpi ulnaris 4 (Medical Research Council, 1942, grading): flexor digitorum profundus 3 to 4th digit and 4 to 5th: all intrinsic muscles paralysed except adductor pollicis and the first dorsal interosseus. Normal sensibility; hypohidrosis in the palmar distribution of the nerve. Electrical reactions and electromyography confirmed the diagnosis of a partial lesion of the ulnar nerve.

There was some derangement of *radial* nerve function, but there seemed to be no doubt that recovery was well advanced.

Contractures: Slight flexor contracture of all digits, but no more than is commonly seen after nerve injuries in the forearm.

Blood-vessels: Radial pulse not palpable, ulnar pulse normal.

The diagnosis seemed clear enough. The median nerve was almost certainly divided at the level of the wound; the radial artery had been damaged at the same level; there was, presumably, a partial division of the ulnar nerve at the site of fracture; and the state of the bones was obvious. There was no suggestion whatever of an ischæmic lesion, and the case was not investigated from this standpoint. The plan of attack on this severely damaged forearm was, however, the subject of some debate; it was finally decided to deal with the non-union of the ulna and with the median nerve lesion at the same session. There was no indication for exploring the ulnar, since the activity of adductor pollicis and the first dorsal interosseus gave promise of spontaneous recovery in the other ulnar intrinsic muscles.

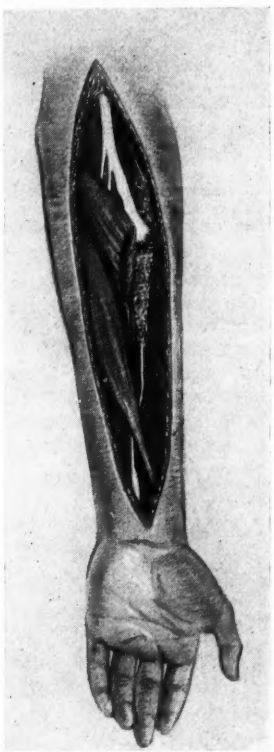

FIG. 319.—The state of affairs at operation. For the sake of clarity the deformity of the forearm is not shown, nor is the scarring properly represented—it was such that pronator teres and flexor carpi radialis were with difficulty recognizable proximally. For description *see* text.

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OPERATION (*Feb.* 2, 1944).—The fracture of the ulna was first exposed, then the divided median nerve. The latter ended in a large neuroma (*Fig.* 319) at a point 9 cm. below the medial epicondyle. The peripheral stump, however, could not be identified until the incision had been carried down to the level of the wrist. Here the nerve was only 4 mm. × 2 mm. in diameter, was purplish-grey in colour, and its bundles were only just visible. On tracing it upwards it was found to become even smaller, diameter 2·5 mm. × 1·5 mm., dirty grey in colour, soft

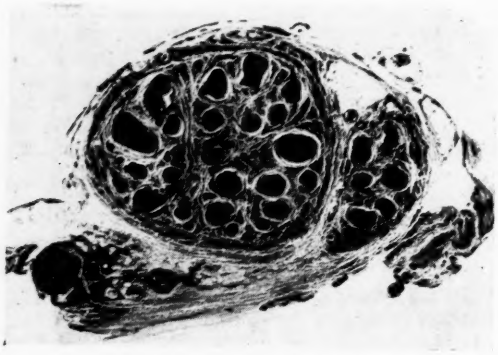

FIG. 320.—A transverse section of the specimen of the distal stump of the median nerve. Masson. (× 12.) The bundles are filled with deeply-staining collagen.

and apparently avascular. This minute thread was lost above in the much damaged forearm muscles. The resemblance to an ischæmic nerve lesion was recorded at the time, though with some hesitation since the neighbouring muscles showed no change apart from those attributable to past sepsis and denervation. The anterior interosseous artery was

FIG. 321.—A specimen of the distal trunk of the median nerve from another case in which the nerve had been completely divided, and had undergone Wallerian degeneration only. Compare with *Fig.* 320; the two sections were stained and photographed together. (× 12.)

nowhere to be seen. Since repair of the nerve was entirely out of the question, a length of the thread-like peripheral stump was removed for examination. It is regrettable that no muscle was removed, but it did not then occur to us that there was any reason for examining muscle, especially as electrical stimulation of the central stump produced contraction in pronator teres, flexor carpi radialis and the long flexors of the fingers, presumably flexor sublimis digitorum. The branches to these muscles were, of course, derived from the central stump; they had evidently been severely damaged in continuity, for they had shown no activity clinically as late as twenty-three months after the injury.

Thus when the operation was concluded there was no more than a hazy notion in our minds that this curious shrinkage of the peripheral stump was the result of ischæmia.

HISTOLOGICAL EXAMINATION OF THE PERIPHERAL STUMP.—At the distal end of the specimen the general arrangement of the nerve-bundles was normal (*Fig.* 320). But in internal structure they were greatly changed, for there had been a massive increase of collagen in the endoneurium, leading in most areas to complete obliteration of the Schwann tubes (*Fig.* 322), in some to a great restriction of their lumina. The collagen in the bundles was composed of dense, deeply-staining strands; consequently there was a marked difference even at low power of magnification between this nerve and one which has undergone Wallerian degeneration (*Fig.* 320; cf. *Fig.* 321, which is a similarly stained transverse section

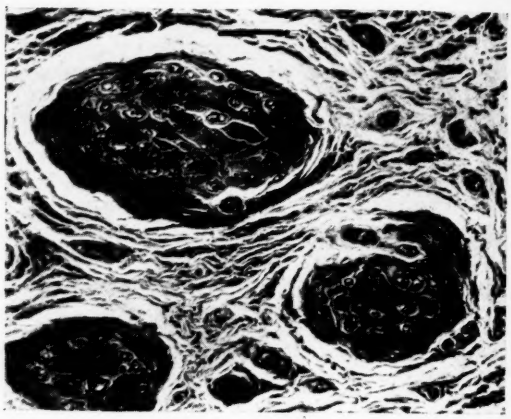

FIG. 322.—Detail from part of the section shown in *Fig.* 320. Masson. Scale = 100μ. The collagenization and hypervascularization of the bundles can be seen.

of the distal trunk of a nerve that had undergone Wallerian degeneration only). Very few Schwann cells could be identified in the bundles, even in those tubes which had not been completely obliterated: at all points in the nerve there was an abnormally large number of small blood-vessels.

At the proximal end of the specimen, where the shrinkage of the nerve was considerable, the internal structure of the bundles was similar to that seen at the distal end, though they were all of smaller diameter, indicating that the shrinkage of the whole trunk was partly due to shrinkage of the individual bundles: the amount of epineurial connective tissue was not significantly different at the two ends. There were also fewer bundles in the proximal than in the distal section, indicating that the shrinkage was also the result of an actual replacement and removal of nerve tissue.

DISCUSSION

Comparison of this specimen with those previously examined (Holmes et al., 1944) showed beyond all reasonable doubt that the state of the peripheral stump was due to ischæmia; the extreme shrinkage of the nerve as a whole and the obliteration of the Schwann tubes by dense collagen is found, so far as we know, only in

nerves damaged by severe ischæmia. The curious feature of the case was the absence of other evidence of ischæmic damage. It must be admitted that the exclusion of ischæmic changes in muscle is much more difficult than the demonstration of their presence, but it may be said with confidence that had such extreme nerve damage been the result of a regional ischæmia the muscle changes would have been unmistakable to even the most casual observer.

One must, therefore, look for vascular damage having a selective action on the median nerve: there is no need to look very far. The median nerve in the forearm derives its main blood-supply from the anterior interosseous artery, and from a few collateral branches. In any nerve occlusion of the collateral branches is harmless (Adams, 1942, 1943) provided that the longitudinal supply is intact, but interference with the main longitudinal anastomoses is definitely harmful (Durward, 1944). In one case described by Holmes and his colleagues—the last in their series—the only demonstrable arterial lesion in a case of ischæmic paralysis of the forearm, with involvement of the median nerve, was occlusion of the distal part of the anterior interosseous artery. But the nerve lesion was a mild one, and almost complete recovery occurred. The nerve was in continuity and there was, therefore, some opportunity for maintenance of the circulation from above. Here, on the contrary, the nerve had been completely severed high in the forearm and thus the distal stump was completely deprived of its proximal blood-supply. The collateral circulation must have been inadequate too, since the radial artery was destroyed in the proximal third of the forearm. This suggests the important conclusion that in mobilization of the distal stump of a median nerve for the repair of a lesion in the upper third of the forearm the anterior interosseous artery should be treated with respect, for it may be the chief source of nourishment of the nerve: perhaps neglect of this precaution accounts for some of the failures after suture at this level, when conditions were not at all adverse in other respects.

SUMMARY

1. A case is described of division of the median nerve in the upper third of the forearm with severe ischæmic damage of the peripheral stump. There was, apparently, no concomitant ischæmia of muscle.

2. The anterior interosseous artery was destroyed, and it is suggested that this, together with the division of the nerve, cut off the main longitudinal blood-supply of the nerve.

3. In the performance of operations on the forearm for repair of proximal division of the median nerve, it is probably wise to avoid disturbance of the anterior interosseous artery.

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A CASE OF TRAUMATIC DIAPHRAGMATIC HERNIA

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TRAUMATIC hernia may be caused by a direct injury to the diaphragm, such as by a knife passing through the ribs and wounding the diaphragm, by necrosis produced by pressure from a drainage tube of an empyema lying on its upper surface, or it may arise from rupture of a subphrenic abscess into the thoracic cavity. On the other hand the condition may be brought about by indirect violence, as by a crushing accident which ruptures the diaphragm and allows the stomach, the intestines, or the spleen to enter the thoracic cavity. Such herniæ have no sac and might therefore more accurately be described as false herniæ or eviscerations. The case here reported belongs to this latter type.

CASE REPORT

CLINICAL HISTORY.—The patient, A. D., a coal miner aged 53, was admitted to the Royal Infirmary in Jan. 27, 1943. He gave a history that three years previously he was crushed by a fall of coal while working in a mine. He was treated in Chirk hospital for twenty-four weeks for fractured femur and fractured upper lumbar spine. After this he recovered completely and went back to work.

In January, 1930, he was knocked down by a horse and was hospitalized for six weeks suffering from a crushed foot. There was no sign or symptom of chest or stomach trouble after either of these accidents.

A third accident occurred in November, 1941, when he was knocked down by a waggon and hurt his ribs and back. Since then he had pain in his left chest, constant in nature, which kept him awake at night so that he had to sit up in bed and was hardly free from pain for longer than half an hour.

About two months before his admission to the Royal Infirmary he began to vomit irregularly, without relation to the taking of food, and on Jan. 19, 1943, he vomited some blood.

A preliminary X-ray examination of the stomach, by Dr. Charles Salt, of Chirk, showed that barium

after passing down the œsophagus went upwards into the thoracic cavity as far as the left clavicle. He was then referred to the Royal Infirmary.

Fig. 323.—Before operation, showing position of stomach in chest. Patient lying on face.

X-RAY EXAMINATION (Dr. R. E. Roberts).—

Feb. 2.—The stomach is seen to occupy a position in the left thorax, its shape being roughly that of a horseshoe with concavity downwards. The cardiac orifice is at the level of the 11th intercostal space, near the middle line, whilst the pylorus lies in the 10th interspace a few inches to the outer side of the cardiac orifice. The body and pyloric antrum, corresponding to the curved portion of the horseshoe, rises into the thorax as high as the 4th rib posteriorly, or the 2nd intercostal space anteriorly. (*Figs.* 323–325.)

The outline of the left diaphragm cannot be made out. The region around the pyloric antrum shows a homogeneous semi-opacity containing a few gas translucencies; the splenic flexure is seen lying to the left side of the stomach, and rising into the thorax as high as the 5th rib in the axillary line.

OPERATION (Feb. 12).—The patient was placed on the operating table on his side, with the left side uppermost. A long incision was made in the chest through the 7th intercostal space and the pleural cavity opened. Rib spreaders were then inserted to keep the ribs apart. The left lung was found to be collapsed and compressed by abdominal contents, which had passed up into the thoracic cavity through a very large rent in the diaphragm. The whole of the stomach was in the chest, being attached below by the œsophagus at the œsophageal hiatus and forming from this point a horseshoe-shaped loop upwards towards the clavicle and downwards to the level of the 7th rib where it joined the dislocated duodenum. The whole of the spleen and the splenic flexure of the colon had also passed through the rent.

No adhesions were present, but it was with some difficulty that the stomach and spleen and colon were gradually reduced into the abdominal cavity.

A search was then made for the edges of the rent, which were found to be widely separated.

Three stay sutures of No. 2 chromic catgut were then inserted in the anterior lip of the rent and three in the posterior lip, and by traction on these sutures

Fig. 324.—Before operation, showing position of stomach in chest. Patient lying on back.

Fig. 325.—Before operation. The fluid level is in the inverted stomach. Patient erect.

an attempt was made to draw the edges together. This could only be effected after first crushing the phrenic nerve with a hæmostat as it lay on the

pericardium so as to paralyse and relax the diaphragm, and by resecting about an inch of the anterior and posterior ends of the 7th and 8th ribs so as to allow the chest wall to fall in towards the middle line and so narrow the opening. With the aid of these manœuvres the stay sutures could be approximated.

With a long pair of scissors the edges of the rent were freshened by separating the pleura on their upper surfaces from the peritoneum on their under surfaces. These two membranes had united with each other to form a smooth lining covering the edges so that no muscular or membranous fibres were visible until the freshening process had been completed. Further interrupted strong (No. 2 chromic) catgut sutures were then inserted between the stay sutures as they held the edges of the rent together, and the remains of the diaphragm were thus sutured together so as to obliterate the rent and reconstruct the diaphragm without undue tension. The sutures were passed on a curved needle from the pleural surface through the fibres of the diaphragm into the peritoneal surface, then from the peritoneal surface of the opposite side through the diaphragm to the pleural surface, and the knot tied on the pleural aspect. It was felt that further reinforcement of the suture line was advisable in case the sutures gave way. Two long strips of fascia lata were therefore excised from the left thigh. These strips were

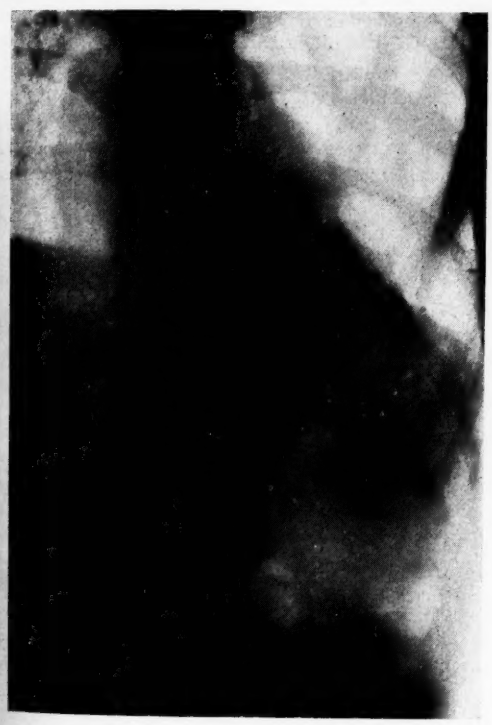

Fig. 326.—March 3, thirty-two days after operation, showing stomach in normal position in the abdomen. Patient erect.

interlaced by means of a Gallie needle along the upper surface of the suture line like bootlaces, passing from the mesial end of the suture line near the œsophageal opening to the lateral end at its junction with the chest wall.

The original incision in the chest wall was then closed by approximating the 7th and 8th ribs and sewing up the intercostal muscles and the muscles of the chest wall. No drainage was used because it was considered that any effusion which might

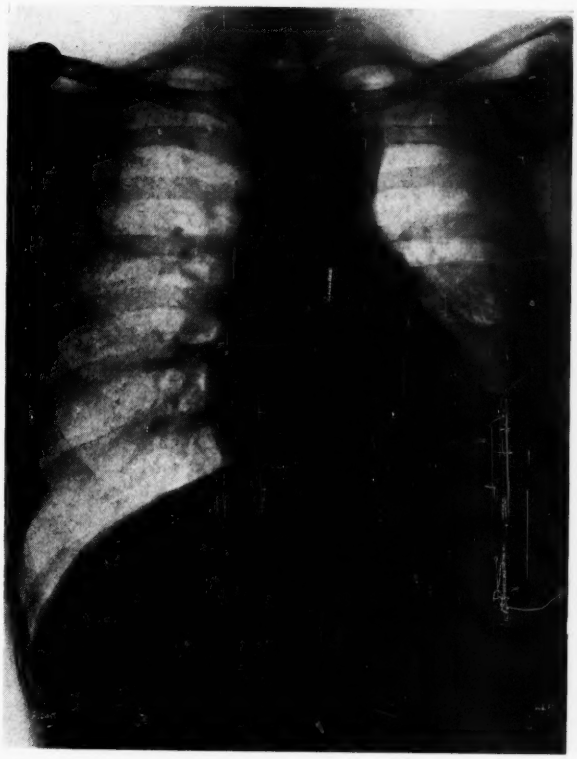

Fig. 327.—Thirty-two days after operation. Lung expanded, fluid disappeared. Patient erect.

subsequently arise could effectively be dealt with by aspiration.

Anæsthesia was carried out by G. O. E., using an intratracheal closed circuit method (Dr. Halton).

The whole operation took one and a half hours. The patient was given a continuous transfusion of blood and left the table in good condition.

PROGRESS.—On Feb. 17 the chest was explored by a needle and 32 oz. of serous fluid were removed from the pleural cavity. Further aspiration was done on Feb. 19 and on Feb. 23, when small amounts of fluid were withdrawn.

On Feb. 25 *X-ray examination* showed no fluid in the chest and the lung was expanding satisfactorily. The patient felt quite comfortable and was able to leave his bed.

March 3.—The stomach is now seen to be completely reduced into the abdomen. Its cardiac end is at the level of the 11th rib posteriorly, being surmounted by a well-defined diaphragm (*Fig.* 326). The pylorus is now at the level of the 2nd lumbar vertebra. The stomach now presents a normal J-shape. The splenic flexure is also in its normal position.

Subsequent X-ray examination showed that the left lung had expanded, and that the residual pleural space and the fluid had disappeared, the bony cage of the left thorax showing a slight falling in. The heart was not appreciably displaced (*Fig.* 327).

Subsequent progress has been uneventful and he is now at work.

AN OPERATION FOR REMOVAL OF CARCINOMA OF THE ŒSOPHAGUS WITH PRESTERNAL ŒSOPHAGO-GASTROSTOMY

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THE treatment of carcinoma of the œsophagus varies according to the situation of the lesion. For the more superficial tumours at the upper end, radiotherapy has become the method of choice, but within the chest it is apparently not possible to destroy a growth completely by this means, without causing fatal damage to the vital tissues of the heart and lungs. Fortunately, the risk of surgical removal of the intrathoracic part of the œsophagus has latterly been very greatly reduced by new methods of anæsthesia and by measures to combat operative shock and post-operative infection. Although the main difficulties of transthoracic œsophagectomy have been largely overcome, however, the restoration of natural swallowing afterwards has only been satisfactorily achieved in the case of growths of the lower part of the organ. Here the tumour can be removed and the stomach mobilized through the diaphragm into the chest to obtain a direct gastro-œsophageal anastomosis in the manner first described by Sauerbruch. This operation has now become almost standardized, and Garlock has recently shown that it can be made to include growths which extend as high as the concavity of the aortic arch by bringing the œsophagus to the left of this structure.

The anastomosis is a precarious one by intestinal standards because of the friable nature and longitudinal direction of the fibres of the bare outer muscular coat of the œsophagus, but by telescoping the cut end into the peritoneal surface of the fundus of the stomach, and by anchoring the stomach to the pleura to relieve the tension on the suture line, a tolerably safe union can be obtained. There is, however, a limit to which this can be done and no comparable way of restoring deglutition after mid-upper œsophageal resection has yet been described. At present the only alternative to Torek's original plan of establishing permanent cervical and epigastric fistulæ is the construction of a skin-tube between them. This is an unsatisfactory position because the fashioning of such a tube requires a number of plastic operations extending over a long period of time after the main operation, and the patient may even develop metastases before the process is complete. Moreover, skin does not tolerate gastric juice well, and many attempts to construct skin tubes have ended in failure. On the other hand, the fistulæ themselves are very troublesome and sore, and it is questionable whether a patient is greatly benefited by the successful removal of his growth unless he is able to swallow his food and saliva fairly soon afterwards.

The same problem of how to close the gap in the alimentary canal arises in the case of a cardiac growth which involves so much of the stomach wall that an endothoracic anastomosis cannot be performed without inadequate removal of tissue. The question in both cases is how to get the stomach to come up to meet the œsophagus, and it is therefore significant that as the patient lies propped up in bed with flexed neck and trunk, the shortest distance between the cricoid and the duodenum lies in the plane of the sternum. For this reason the stomach can be mobilized higher in front of the sternum than round the curve of the posterior mediastinum behind the heart. In this paper an operation is therefore suggested in which an œsophageal or gastro-œsophageal resection of a growth is followed by a presternal or suprasternal anastomosis between the stump of the œsophagus and the stomach or the remaining part of it (*Fig.* 328).

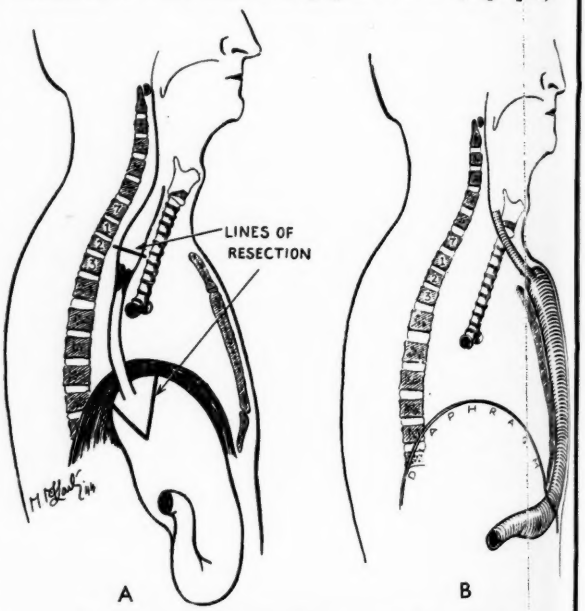

FIG. 328.—Diagramatic representation of the operation. A Shows the gastro-œsophageal resection; B, Shows the remainder of the stomach brought upwards to the front of the sternum and anastomosed to the stump of the œsophagus.

Such a procedure is, of course, a formidable undertaking, but this is a consideration which must be viewed against the hopeless and distressing alternative for a patient with a growth of the œsophagus or cardia.

The operation has been put into practice in three cases, with one death from accidental pneumothorax of the opposite side of the chest,

one death from empyema after eight months, and one patient alive, taking soft food by mouth and enjoying her life at home eighteen months later. The following is a description of the operation as I have performed it, with such modifications as experience has suggested.

TECHNIQUE

Pre-operative preparation consists in rest in bed for two to four weeks with a high calorie fluid diet by mouth, unless obstruction is absolute. This period is used to remove all septic foci in the mouth, to reduce œsophageal stasis by regular lavage with an œsophageal tube, and to give a blood transfusion if required. Œsophagoscopy is essential to confirm the diagnosis by inspection and biopsy. It may also provide evidence of fixity of the gullet in the mediastinum or, with bronchoscopy, involvement of the trachea or left bronchus, which would, of course, contra-indicate operation. At the same time it affords an opportunity of dilating the stricture if necessary, to facilitate feeding and œsophageal drainage. Shortly before operation a course of local chemotherapy is instituted in which the lavage after each feed is followed by a dose of sulphathiazol suspension in order to reduce the infection of the growth. Breathing exercises are instituted and the patient is familiarized with the oxygen mask in preparation for the post-operative period. Preliminary jejunostomy is not necessary if there has been a good response to these measures, but may be required if the œsophageal obstruction has not sufficiently diminished under treatment.

Cyclopropane and oxygen administered by cuffed endotracheal tube is the method of anæsthesia adopted, in order to give the anæsthetist unhampered control of respiration by means of positive pressure when the chest is open and the physiological mechanism for expanding the lung cannot function. The operation commences with the setting up of a drip transfusion of blood, followed later by plasma, and in anticipation of a lengthy dissection measures to keep the patient warm are taken from the outset, and pulse-rate and blood-pressure readings are taken at 15-minute intervals.

The procedure for a growth in the upper half or two-thirds of the œsophagus will first be described. For this, a right thoracotomy is chosen in order that the dissection in the region of the aortic arch may proceed more readily under vision. The patient is therefore placed in a half-left lateral position and his right arm is supported by an assistant, as it will be required to be moved during the operation. The right pleural cavity is opened through the bed of the 6th rib and any parietal adhesions of the lung are separated until it lies on the mediastinum and the growth in the œsophagus is exposed. If there is no obvious infiltration of the mediastinum the pleural covering is incised, the azygos vein is divided, and the growth is gently dissected free, care being taken to avoid tearing the left pleura below. Even with positive pressure in the trachea such a tear may have a valvular action, and if it occurs there is danger of a contralateral pneumothorax, with obstruction of the pulmonary blood-flow, and general circulatory failure (*see Case* 3). The opening should therefore be carefully sutured, but if this is not possible a vent tube should be put through it until the end of the operation, to prevent any possibility of valve action and to allow the anæsthetist to keep the lung fully expanded.

While the mediastinal dissection is proceeding, in fact, as soon as it has been decided that the condition is operable, an independent assistant makes two other short incisions. The first, about 1½ in. long, is made into the peritoneal cavity through the midline of the epigastrium and the wound is covered with a towel until it is required. The second incision, about 1 in. long, is made transversely in the skin over the suprasternal notch. Through this opening the assistant tunnels downwards in the relatively bloodless subcutaneous tissue in front of the sternum, by the method of repeated scissor-spreading, as far as the length of the scissors will allow. This tunnel will eventually accommodate the upper stump of the œsophagus, and if it is prepared in advance in this way much time will be saved in the later manœuvre.

Meanwhile, within the chest, the surgeon completes the mobilization of the whole length of the œsophagus by freeing the two ends. Below, the attachments of the cardia are separated from the œsophageal hiatus of the diaphragm, the peritoneal cavity being deliberately opened anteriorly. Above, the loose cellular tissue at the root of the neck strips easily from the œsophagus until the cricoid is reached. Great gentleness is required here to avoid damage to the important descending œsophageal branches of the inferior thyroid arteries. When the œsophagus is quite free from the cricoid to the stomach, a special long curved clamp is introduced into the epigastric incision and, passing between the stomach and the left lobe of the liver, is guided by the surgeon's fingers through the peritoneal opening which was made at the œsophageal hiatus of the diaphragm, and continuing high into the chest is clamped across the œsophagus above the growth. The first two fingers of the surgeon's left hand now slide up the œsophagus to the cricoid, and work their way forward in the neck between the pharynx and the right carotid sheath until they present at the assistant's suprasternal incision which is also the mouth of the subcutaneous tunnel. This manœuvre is illustrated in the diagram for greater clarity (*Fig.* 329). The fingers guide the blades of a second clamp from the cervical incision down into the chest, where they are applied to the œsophagus immediately above the first clamp. The œsophagus is then divided between the clamps, which are immediately withdrawn to the exterior, carrying the two parts of the œsophagus with them.

The lower part is carried through the diaphragm and out of the epigastric incision ; the upper part is drawn through the root of the neck to the space

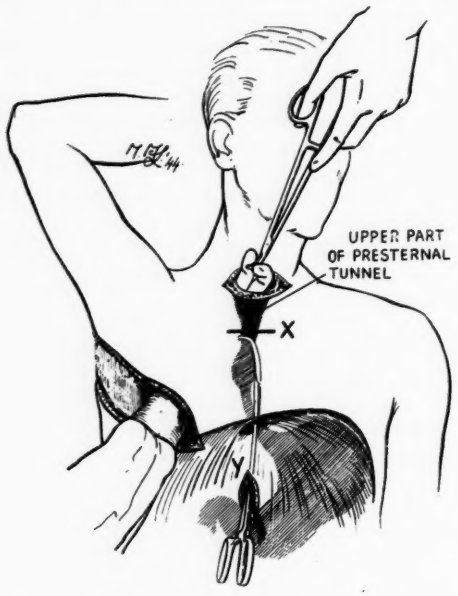

Fig. 329.—The surgeon's fingers protruding from the suprasternal incision (*see* text).

of Burns, whence it is thrust down the presternal tunnel as far as it will go. The assistant completes the cervical part of the operation by bringing the end of the upper part of the œsophagus out through a short counter-incision over the tunnel, after which the suprasternal incision, having served its purpose, is sutured. The edges of the œsophageal opening in the disphragm may be approximated if necessary, after which the mediastinum is dusted with sulphonamide powder and the thorax is closed with under-water drainage. Dressings are applied, the lung is expanded by the anæsthetist, and the patient is slowly placed on his back.

The first stage of the operation having now been completed, the general condition of the patient is critically reviewed by the surgeon and the anæsthetist, to decide whether it is possible to proceed at once to the mobilization of the stomach into the presternal position. If the anæsthetist's chart indicates a progressive fall of blood-pressure, especially if the systolic reading is below 100 mm. of mercury, the second stage should be postponed until a later date. In case of doubt, the patient's response to an interval of ten-minutes' rest is a useful indication of the degree of reserve vitality he possesses.

If it is considered wiser to desist, the upper end of the œsophagus is sutured to the opening in the presternal tunnel, the growth is removed from the lower œsophagus and this end is sutured to the skin of the epigastrium. In the interval before the second stage the patient will be fed through the lower œsophagostomy and later the two fistulæ may be temporarily connected by an external tube, as in Torek's case.

The second stage, can, however, be fairly rapidly performed and it is very desirable to avoid the epigastric fistula if possible. If therefore, it is decided to proceed at once to the anastomosis, the epigastric incision is enlarged and the presternal subcutaneous tunnel is extended downwards to join it. The stomach is now mobilized by gentle traction on the œsophagus which facilitates the division of the root of the coronary vessels beyond the glands in this region, as well as the vasa brevia to the spleen, and the great omentum along the greater curve. This can be done without visible impairment of the blood-supply of the fundus of the stomach, if care has been taken to avoid damage to the pyloric and right gastric-epiploic vessels. It will now be found that the stomach can be withdrawn entirely from the abdomen and laid on the front of the chest with the fundus as high as the sternal angle of Ludwig. By mobilizing the duodenum as well, it can be drawn even higher so that the pylorus lies over the xiphisternum and the fundus reaches above the clavicle. This degree of mobilization is only necessary after a very high resection of the œsophagus, but enough of the stomach is freed to allow the fundus to reach the upper end of the œsophagus without tension. The growth, with the lower œsophagus, the cardia, the upper part of the lesser curve, and the glands which have been dissected up from the cœliac axis are now removed and the opening in the stomach is closed in two layers. The fundus is drawn up the presternal tunnel to the counter incision for the anastomosis, while the assistant closes the epigastric wound, taking care to leave plenty of room for the stomach to pass between the muscles without constriction. The fundus is sutured to the chest wall about 1 in. higher than the upper end of the œsophagus, which is laid on its surface and sutured in position. A small incision is now made into the stomach wall opposite the end of the œsophagus and an anastomosis is made between the two openings, the suture line being covered by drawing the anterior wall of stomach up over it. The skin over the anastomosis is closed with drainage of the subcutaneous tissue and the operation is completed by passing a small nasal tube through the anastomosis into the presternal part of the stomach.

When the operation is performed for growths of the lower part of the œsophagus or of the cardiac end of the stomach, the exposure is through the bed of the seventh *left* rib, the diaphragm is incised radially from the œsophageal hiatus, and the growth is dissected free. The first steps are therefore those of Sauerbruch's operation, which will be the method of choice if the growth is limited to the cardia or does not extend higher than the concavity of the aortic arch. The surgeon may well defer the selection of an intra- or an extra-thoracic anastomosis

until, with the parts in his hands, he is in a position to assess the practicability of the former. If this is not possible he need not abandon the resection, but may proceed to apply the principle of the operation described above with sufficient modification to conform to the anatomy of the aortic arch. Thus the œsophagus must be freed by blunt dissection with the finger between the deep side of the arch and the azygos vein after infiltrating this region with ½ per cent novocain; the dissection of the root of the neck is to the left of the trachea, and in passing the œsophageal clamp down from the neck into the chest it must follow the same path between azygos vein and aortic arch before being applied to the lower œsophagus and withdrawn. The blood-supply of the œsophagus is more precarious when it is divided low down, and particular care must be taken in dissecting the root of the neck not to damage the vessels joining the upper end from the inferior thyroid trunks. It is expedient also to extend the incision across the costal margin and left rectus muscle to the epigastrium to obtain a wide thoracico-abdominal exposure for the dissection of an extensive growth in the stomach. The spleen and proximal omentum should be removed with the growth and the stomach divided about half-way down the greater curvature and as far as the incisura on the lesser curve. *Case* 3 shows that this degree of removal of tissue is still compatible with a presternal anastomosis if the duodenum is mobilized as well as the stomach. Apart from the approach and exposure, and the dissection below the diaphragm and opposite the aortic arch, the execution of the operation follows the same lines as that described for upper œsophageal resection.

During the post-operative period the patient is sustained by fluid diet by the nasal tube, through which sulphathiazol suspension is also administered. When not used for this purpose the tube is kept open to the atmosphere to prevent gas distension of the stomach and thus remove any internal strain on the anastomosis. Leakage here is fraught with danger of widespread infection of the subcutaneous tissue in front of the chest and a careful watch must be kept to prevent this. The integrity of the suture line may be tested by a drink of methylene blue which will ooze from the local drainage tubes if it is not sound. Continuous suction to remove escaped saliva may prevent a complete breakdown, but if inflammation is spreading it is better to limit this by opening up the wound and intubating the fistula.

If the anastomosis holds, nothing is allowed by mouth until the œsophagus and stomach are firmly adherent in their new position, but mouth-washes may be freely used. Sips of fluid may be given on the seventh day and the nasal tube is removed on the tenth day if all is well. The under-water drain is removed when the discharge has ceased to be copious, but the residual pleural fluid is aspirated at intervals and cultured, and penicillin may be injected at the same time to minimize the risk of secondary infection from the bronchopneumonic lung.

CASE REPORTS

Case 1.—The patient was a man of 61 with chronic bronchitis, an emphysematous chest, and a squamous carcinoma 2½ in. long in the middle of the œsophagus. With Mr. C. B. Lewis administering the anæsthetic, the operation was performed on July 24, 1942, through a right thoracotomy incision. Widespread adhesions of the lung and inflammation of the mediastinum made the dissection lengthy and difficult, and after removal of the growth it was decided to postpone the mobilization of the stomach to a later date. The upper œsophagus was therefore left as a fistula near the suprasternal notch, while the lower œsophagus was brought to the surface of the epigastrium. Post-operative bronchopneumonia with gangrene of the right lower lobe resulted in an empyema which was drained, but later required a partial thoracoplasty to obliterate the cavity. The patient improved after this, but he was much troubled by persistent regurgitation from his lower fistula and he failed to put on weight. Eight months after the œsophagectomy it was therefore decided to proceed to the mobilization of the stomach into the presternal position. During this procedure a second empyema was found presenting at the œsophageal hiatus of the diaphragm, where it was closed off below by the spleen and stump of the œsophagus. This was evacuated and drained from below and the presternal operation was completed, but the patient died a week later.

The post-mortem showed that the mediastinal empyema connected with a necrotic area of the inner surface of the lower lobe of the right lung. There was no secondary growth in the mediastinum and the stomach had not sloughed in its new position, but was adherent by fibrinous tissue to the skin and the front of the chest wall.

Case 2.—This case was more encouraging; the patient was a woman aged 60 who had lost 4½ st. in weight and suffered from dysphagia due to a growth in the lower part of the œsophagus, the upper edge of which was 3 in. above the cardia. Her operation was performed on April 2, 1943, the anæsthetic being given by Mr. Stout. A right thoracotomy was again chosen, the lower 5 in. of œsophagus were removed together with a fringe of gastric mucosa, and the complete operation of presternal œsophago-gastrostomy was performed uneventfully in one stage. Bronchopneumonia with empyema again supervened, but cleared up after drainage and healed during the next four months. The anastomosis broke down six days after operation and formed a fistula about 1 in. across, with upper and lower openings. These were connected by an indwelling plastic tube through which the patient was able to take liquid or soft solid foods by mouth.

The specimen was reported as a columnar-celled adenocarcinoma by Dr. W. W. Woods, although at operation it had not appeared to extend to the cardia. Seven months after the œsophagectomy the fistula was explored and some nodules of growth were found in the gastric mucosa, with a metastatic gland on the lesser curve. The whole stomach was therefore mobilized once more and a partial gastrectomy was performed from the cardiac end, the line of section being two-thirds the distance along the lesser curve and one-third the distance along the greater curve.

It was surprising to find that even now a direct anastomosis could be made without tension between the end of the œsophagus and the greater curve of the stomach. Unfortunately post-operative infection of the presternal cellular tissue caused a further breakdown of the anastomosis which healed but left a gap of 3 in. between the œsophageal and gastric openings.

This patient has been discharged home with an indwelling plastic tube across the fistula; she lives a fairly normal life, taking soft foods by mouth and has put on 19lb. in weight. She removes the tube at night and replaces it in the morning in order to keep it fairly watertight. If the tube gets blocked she takes it out, cleans it and puts in back, though this is seldom necessary. She has no trouble with regurgitation into her mouth and the disability of the fistula is not at present sufficient to warrant a further operation. She is, however, unable to exert herself, as this causes the fistula to leak. Full of courage, she proposes to come into hospital for plastic closure of the fistula.

Case 3.—The third patient was a fairly healthy man of 48 with three months' history of lassitude, loss of weight, and difficulty in swallowing solid foods due to a columnar-celled carcinoma of the cardia. His operation was performed on Feb. 29, 1944, cyclopropane and oxygen being administered by Dr. Parry Brown. Through a left abdomino-thoracic incision the growth was found to extend right across the fundus of the stomach and 1½ in. up the œsophagus, which opened into the base of a large malignant crater. There was one indurated œsophageal lymph-node, several outside the cardia, and a mass of glands around the root of the coronary artery, but the growth itself had not broken through the outer coat of œsophagus or stomach. The mass was too extensive to permit of endothoracic anastomosis, but after resection from 3 in. above the cardia to a line joining the incisura and the middle of the greater curve of the stomach, a presternal œsophago-gastrostomy was arranged without tension. A satisfactory excision of the growth and glands was obtained, but in spite of transfusion and high oxygen concentration in the respiratory gas a progressive circulatory collapse developed and the patient died two hours after the operation.

Post-mortem examination showed a small tear ½ in. long in the right mediastinal pleura with a partial right pneumothorax. Air had been able to enter but not to escape from the right side of the chest, and although partial inflation of the lung had been maintained by positive pressure, the pulmonary blood-flow had been gravely constricted and collapse of the general circulation had followed.

DISCUSSION

These cases were to some extent experimental, but although they were in many ways disappointing, they do indicate the general feasibility of the plan of operation as well as some of the difficulties and dangers that may be encountered. Naturally, there is much to be learnt in the further development of the technique, but it is hoped that the method may prove a step forward in the treatment of carcinoma in this region, since it offers immediate restoration of swallowing if primary healing is obtained. On the other hand, it is noteworthy that breakdown of the anastomosis is not fatal as it would be inside the thorax, and that the fistula which results is easily controlled by a short indwelling tube and may be repaired at leisure by a further adjustment of the stomach upwards.

The reduction of blood-supply involved in transposing the viscera into the presternal position position is apparently not incompatible with their survival. In neither of the two cases in which the whole stomach was mobilized was any change observed in the colour of the fundus, and, in fact, no necrosis occurred post-operatively. Kirschner made similar observations in 1920 in utilizing the same degree of mobilization of the stomach to by-pass a simple stricture of the œsophagus in 5 cases. He found no evidence of deficient circulation at operation, and post-mortem examination of 4 patients who died showed no sloughing of the stomach, although one contained an acute ulcer. The patient who survived was observed for two years, during which time she ate a full diet and had a normal digestion. My second case also has no disturbance in this way, although her food has to be finely divided.

The blood-supply of the transposed œsophagus is derived from the descending œsophageal branches of the inferior thyroid vessels. These are apparently able to maintain a sufficient blood-flow to the lower middle œsophagus on account of the longitudinal direction of the intrinsic vessels in the œsophageal wall, but for this reason it is important to avoid trauma to the gullet and its superior connexions during the dissection. Doubts on the viability of the lower œsophagus when separated from its inferior vessels have mostly been expressed to explain the breakdown of an endothoracic anastomosis, but other factors are enough to account for this. In the 3 cases described, the blood-supply to the œsophagus was, in fact, adequate; *Case* 1 was a high resection with only a short upper œsophageal stump, but in the other 2 cases the gullet was divided 5 in. and 3 in. from the cardia respectively. It survived uneventfully in the former case and although the last patient did not recover from the operation, the end of the œsophagus was observed to ooze when it was divided above the clamp.

The greatest danger to the patient after recovery from operative shock is that of infection of the pleural effusion from the bronchopneumonic lung. I have not yet had the opportunity of injecting penicillin into the pleural cavity after aspirating the fluid, but its effectiveness in early empyemata suggests that this may be a valuable preventive measure.

Although the operation was primarily designed to obviate the necessity for a skin tube after mid-upper œsophageal resection, its applicability to the lower œsophagus and cardia gives the surgeon greater freedom of action when exploring a growth here. His aim must be to perform an intrathoracic resection and anastomosis if possible, since it involves so much less anatomical disturbance, but he may find that the extrathoracic

method described is necessary to secure complete removal of the type of growth that spreads longitudinally up the œsophagus or widely into the stomach.

Technical feasibility is, however, not the only factor involved in the choice of method. As experience grows, the relative mortality, post-operative disability, and the incidence of recurrence of growth will become of increasing importance. These may be left to the future, but it is worth noting that a gastro-œsophageal junction immediately under the skin has certain advantages. Thus it may be manipulated if food is held up there, or it may be compressed if regurgitation due to lack of a cardia is troublesome, as it tends to be with an intrathoracic anastomosis. More important perhaps is the fact that it can be palpated periodically for recurrent growth, which would then be accessible to X-ray treatment. Alternatively, it may well be that routine post-operative radiotherapy to the superficial stomach and œsophagus would give better long-term results in this form of cancer than are obtainable by operation alone.

I should like to thank the Royal Society of Medicine for allowing me to demonstrate *Case* 2 at a recent meeting of the Clinical Section, and especially to thank my assistants and anæsthetists and the Sisters and Nurses of the London Hospital for their untiring skill and devotion in the care of these patients.

BILATERAL RENAL CARCINOMA

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BILATERAL renal carcinoma is a very rare finding, only about 9 cases being recorded in the literature. In reporting these, the various authors set different standards for accepting the few published cases, but nearly all agree that the diagnosis must eventually be confirmed by post-mortem examination, since the radiological findings may be deceptive.

Most of the authors present their cases as examples of two primary growths, the evidence used against one being a metastasis from the other varying in individual cases.

If there are no other metastases in the body and the renal veins are not involved, it is argued that the likelihood of a solitary secondary deposit in the opposite kidney is remote. Certainly in some case-histories there is such a long interval of time between the discovery of the two growths that there is strong evidence for supposing them to be of separate origin.

However, this question would seem to be idle speculation in most cases, since proof either way is not available.

All but one of the recorded cases are 'hypernephroid' carcinomas or Grawitz tumours, the exception being a bilateral papillary carcinoma of the renal pelves.

SUMMARY OF THE LITERATURE

The first example of a bilateral hypernephroma was recorded by Chute in 1910. The tumours were found post mortem, and there was a secondary deposit in the gut.

In 1914 Parkes Weber described a case of bilateral "malignant hypernephroma" or "adrenal carcinoma". He states that "doubtless the primary growth in the present case was suprarenal". However, this is accepted as an example of bilateral renal hypernephroid carcinoma by some of the other authors. Unfortunately no microphotographs are reproduced, but the description of the gross specimens would seem to indicate an adrenal rather than a renal origin, and I am informed in a personal communication from the author that he still considers his case to have been a bilateral primary adrenal carcinoma, not a renal 'hypernephroid' carcinoma. Parkes Weber's case is, therefore, not included in the total of nine. In 1927 Kinney described a case where the diagnosis was made by retrograde pyelography, but confirmation was never obtained by autopsy examination, and for this reason it is not accepted by the more recent authors, notably Beilin and Neiman. Hunt's case in 1927 is quite straightforward. Nephrectomy was performed for "hypernephroma", and when the patient died, a similar growth was found on the other side; no metastases were found.

Sanford in 1931 performed a nephrectomy for a papillary carcinoma of the renal pelvis, although the other side was suspect radiologically. After death a full post-mortem examination was not allowed, but the remaining kidney was obtained and showed a similar papillary carcinoma. In the absence of radiological evidence of lung metastases he assumed that there were none present.

In 1935 Spengler and Bohrod described 2 cases. The first was diagnosed before death and confirmed by autopsy examination. No secondary deposits were found. In the second instance the tumours were found post mortem, together with multiple secondary deposits in the lung, liver, pancreas, and adrenals, while the right renal vein was filled with tumour tissue.

In 1940 Elward and Spire found bilateral hypernephroma masses after death in a case of

mediastinal neoplasm which had been treated by deep therapy. There were no renal symptoms.

In June, 1942, Forsythe recorded a case of nephrectomy for hypernephroma, followed by the finding of a similar growth on the other side post mortem. He also records a Mallory case-report No. 22091 in 1936, where nephrectomy was followed by the finding of a hypernephroma in the remaining kidney after death 13 years later. There were no metastases. I have been unable to verify this case personally.

In December, 1942, Beilin and Neiman described their case, confirmed by post-mortem examination 103 months after the original nephrectomy for hypernephroma.

These authors quote Siebke (1926) as having described 2 cases, but I am unable to confirm this from the reference given.

The same authors quote Lubarsch as describing 1 case, but on referring to the original paper, I find that Lubarsch includes 1 case of bilateral renal carcinoma in a large series of renal neoplasms which he is reviewing; no details or further references are given. He has seen no case of bilateral hypernephroma, so we may conclude that the case he refers to was a papillary carcinoma. In the absence of clinical data it is not included in the total of recorded cases.

This makes a total of 9 cases of proved bilateral renal carcinoma in the literature, with Kenny's case as a possible tenth.

A further case of bilateral renal carcinoma of the hypernephroma type is now presented.* The provisional diagnosis was made radiologically and was subsequently proved at post mortem.

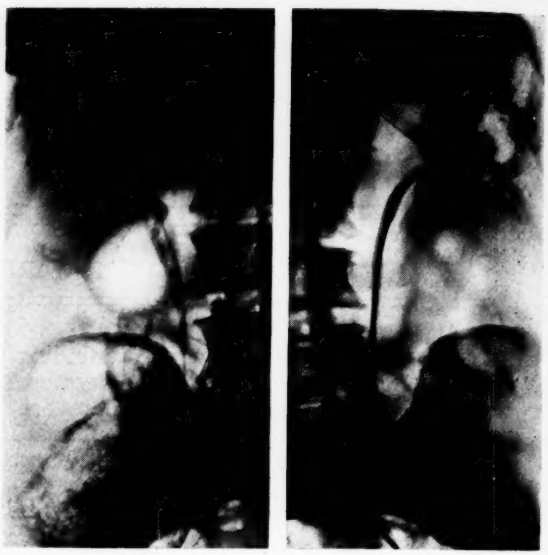
Owing to the absence of positive proof, it is not claimed that this was an example of two primary renal growths, although there is reason for supposing it may have been, especially when compared with the previously accepted cases.

CASE REPORT

J. R., an alert, active, old-age pensioner of 73 years, with no previous or family history of renal disease, was first seen in out-patients in March, 1943, complaining of occasional blood in his urine during the previous three weeks. He had been losing much weight and complained of a painful lump in his right loin, which clinically was a large kidney tumour; his urine contained bright red blood, but was sterile on culture. Other than bronchitic signs in his chest, a general examination revealed no gross pathology. On admission to hospital, intravenous urography showed no excretion from the right kidney up to one and a half hours, while visualization of the left kidney was also very poor. Cystoscopy was performed, when bright blood was seen issuing from the right ureteric orifice; the left efflux was clear.

Owing to the poor visualization of the left kidney in the excretion urogram, a left ascending pyelogram was deemed necessary before performing a right nephrectomy. Most unexpectedly this showed complete amputation of the upper group of calices, the appearance being that of a neoplasm or cyst (*Fig.* 330, B). This left kidney was neither tender nor palpable, while the urine collected from the ureteric catheter showed a few red blood-corpuscles but no other abnormality. A provisional diagnosis of left hypernephroid carcinoma was made.

A week later a right ascending pyelogram was performed under X-ray screen control but no filling of any calices could be obtained (*Fig.* 333, A). Blood was still passing down this ureter, and thus with the provisional diagnosis of bilateral renal carcinoma the man was discharged home to be seen regularly in the out-patient department.



A B

FIG. 330.—A, Right ascending pyelogram. No filling of renal pelvis could be obtained. B, Left ascending pyelogram, showing complete amputation of upper group of calices.

The hæmaturia decreased and even ceased completely for days at a time, but he was readmitted two months later with an acute bronchitis. His renal function was found to be unaltered, and a repeat chest radiograph still showed no evidence of intrathoracic secondary deposits. The lower pole of his left kidney was now just palpable, while the right kidney was much larger and more fixed.

The bronchitis subsided quickly and he was again under observation in out-patients for a period of eight months, during which time the hæmaturia and bronchitis were his chief symptoms. The left kidney was slowly enlarging.

He was readmitted in November, 1943, with bronchitis, but his general condition had deteriorated considerably. He had lost weight, was bleeding continuously, and both kidneys were painful, necessitating morphia to control the spasms at times.

During this period a large left varicocele developed but caused little discomfort. He became steadily weaker and died in December, 1943, ten months after the onset of symptoms.

SUMMARY OF SPECIAL INVESTIGATIONS.—

Radiography.—March 13: Well-marked Paget's disease in pelvis and lumbar spine. *Excretion urography*—virtually no excretion from *right* kidney up

* This patient was demonstrated at the Urological Section of the Royal Society of Medicine on Nov. 25, 1943. The gross specimen was shown at the Surgical Section of the Royal Society of Medicine on Jan. 5, 1944.

to 1½ hours. *Left*—very poor excretion and visualization. *Left ascending pyelogram*—amputation of upper group of calices due to neoplasm or cyst. *Chest*—no evidence of metastases.

March 22: *Right ascending pyelogram*—lack of filling of any calices.

Nov. 12: *Chest*—no evidence of intrathoracic metastases. *Spine, pelvis, leg bones, skull*—no evidence of metastases. Paget's disease—spine and pelvis.

Nov. 13: R.B.C. +++, pus cells +, casts 0, coliform bacilli +.

Blood.—

March 17: R.B.C. 4,130,000 per c.mm., hæmoglobin 80 per cent, C.I. 1·0.

Nov. 22: R.B.C. 5,200,000 per c.mm., hæmoglobin 62 per cent, C.I. 0·6.

Nov. 12: *Serum phosphatase*—alkaline 8·2 units per 100 ml; acid 2·4 units per 100 ml.

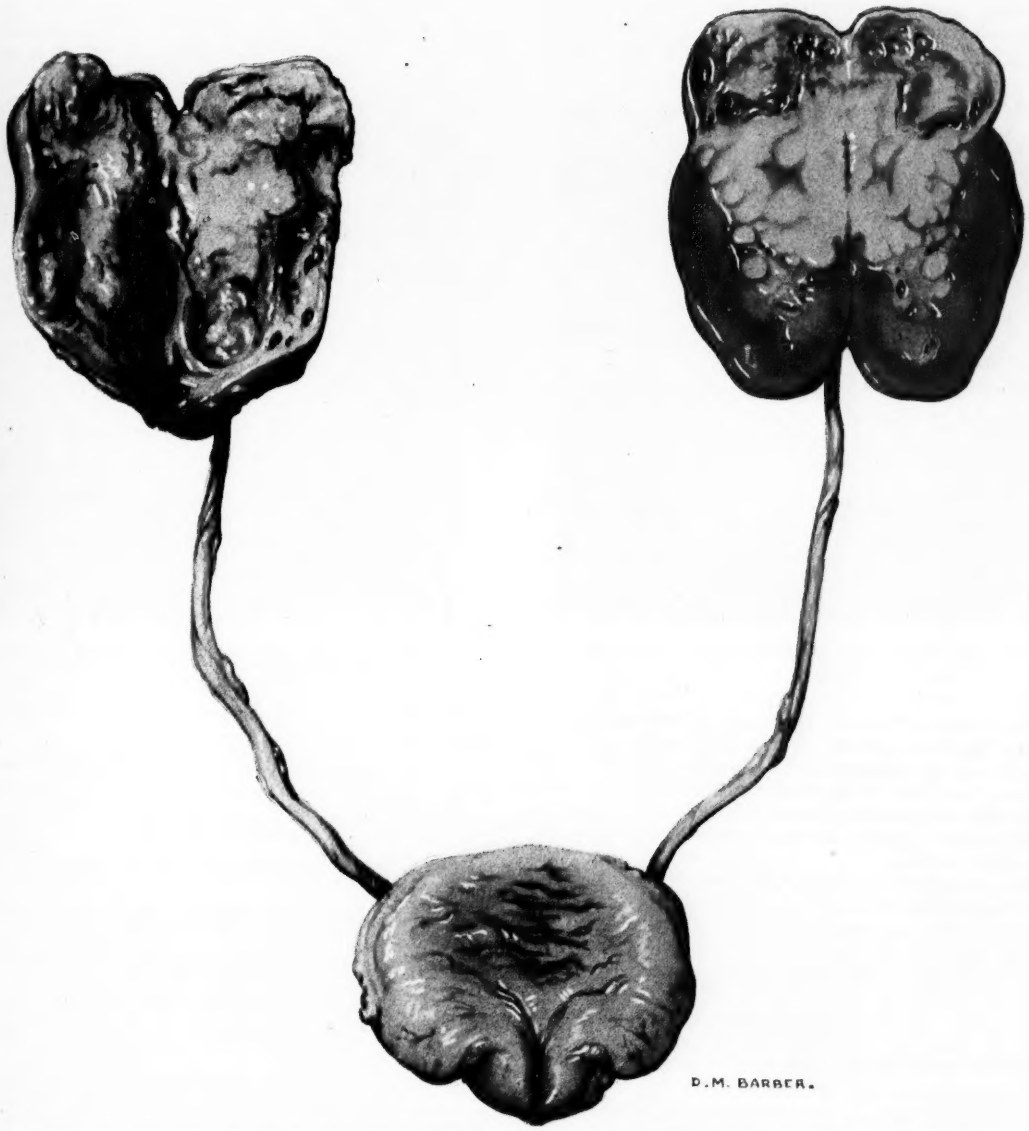


FIG. 331.—Gross specimen showing a typical 'hypernephroma' in the upper half of the left kidney, and a shapeless mass of necrotic growth filling up the right kidney.

Renal Function.—

March 16 :	*Blood-urea*	45	mg.	per	cent
May 29 :	,,	,,	43 mg.	,,	,,
Nov. 12 :	,,	,,	56 mg.	,,	,,
Dec. 10 :	,,	,,	54 mg.	,,	,,

Urine.—

March 13: R.B.C. ++++, pus cells +, casts 0, organisms 0.

Wassermann and Kahn.—Negative.

Blood-pressure.—160/85.

Pulse and Temperature.—Normal throughout.

AT AUTOPSY.—A post-mortem examination performed by Dr. Bray confirmed the diagnosis of bilateral renal neoplasms. Several small nodules on the parietal pleura, which subsequently proved to be secondary hypernephroma deposits, were the only evidence of general metastases.

PATHOLOGY.—The *macroscopical* appearance of the two kidneys and bladder is shown in *Fig.* 331. The *left kidney* contained a typical 'hypernephroma' in the upper pole with normal tissue below it. The *right kidney*, greatly enlarged and shapeless, contained much old and recent blood-clot and collapsed to half its size when cut open. There was a firm, organized, cone-shaped clot blocking the pelvi-ureteric junction while the majority of the dilated sac contained a yellow necrotic papilliferous mass of tissue, nowhere resembling a 'hypernephroma'. There was no evidence of extension along either renal veins, in spite of the recent onset of a varicocele on the left side.

There was no evidence of growth extending along either renal veins, but there were several small metastatic nodules on the parietal pleura. These were not shown in repeated radiographs of the chest.

3. No special claim is made that the case is an example of two primary growths, although similar cases in the literature have been accepted as such.

I would like to express my gratitude to Professor J. Henry Dible for his opinion on the pathological specimen and his kind help with the slides and microphotographs.

I am also grateful to my colleague, Mr. L. Fatti, for his help with the German literature.

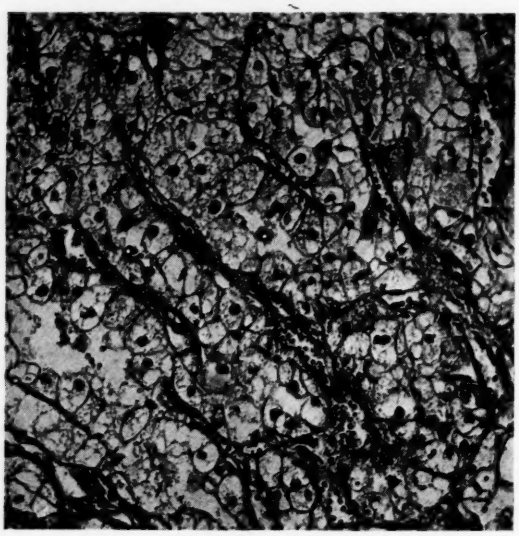


FIG. 332.—Microphotograph of section from left kidney tumour. (x 150.)

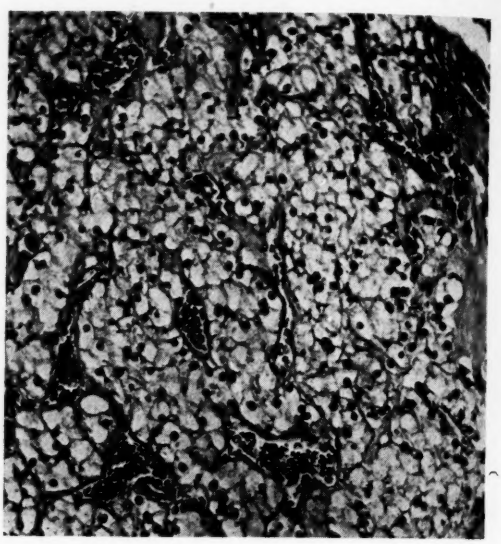


FIG. 333.—Microphotograph from growing edge of necrotic area in right kidney. (× 150.)

Microscopy.—Sections from the left kidney showed typical 'hypernephroma' tissue (*Fig.* 332).

Professor J. Henry Dible, who selected and examined sections from the right kidney for me, reports that two distinct types of cells could be seen, a papilliferous area, and the more typical 'hypernephroma' tissue.

The necrotic material from the distended pelvis showed a papilliferous structure in the areas where any histology could be made out, but a section from the growing edge of this same tissue showed the typical 'hypernephroma' cells (*Fig.* 333).

SUMMARY

1. The previously reported cases of bilateral renal carcinomas are reviewed.

2. A further case is added to the literature, making ten in all. The provisional diagnosis was made radiologically before death, and was subsequently confirmed by post-mortem examination.

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TRAUMATIC ISCHÆMIA OF PERIPHERAL NERVES
WITH SOME OBSERVATIONS ON VOLKMANN'S ISCHÆMIC CONTRACTURE

By ATHOL R. PARKES

SURGEON-IN-CHARGE, PERIPHERAL NERVE INJURIES UNIT SCOTTISH E.M.S.

A NUMBER of cases of injury to a limb complicated by multiple nerve lesions has been seen at a Peripheral Nerve Injuries Centre in Scotland. The aetiology of the nerve lesions was at first obscure, but the conclusion has now been reached that they are due to ischæmia of the nerve trunks, the main factor in the production of which was in most cases pressure of extravasated blood and tissue fluids beneath the deep fascia of the limb.

CASE RECORDS

Most of the patients had been injured some months before their first attendance at the Centre.

LOWER LIMB

Case No. 1.—D. Y., aged 21.

HISTORY.—This naval officer was kicked on the leg while playing football and sustained a simple transverse fracture of the tibia and fibula about

Fig. 334.—Chart showing sensory loss in the distal part of the distribution of musculocutaneous, anterior tibial, posterior tibial, and sural nerves. Note extension of sensitivity to pin-prick along sural distribution.

midshaft. Within a few hours the leg became very swollen and tense, and because of this plaster was not immediately applied. About twelve hours after the injury the patient noticed numbness of the great toe, and this numbness gradually spread centripetally until the whole foot felt 'dead.' The notes state that the posterior tibial pulse at the ankle was not palpable at this time. After five days of elevation of the limb a walking plaster was applied.

EXAMINATION (5 months after injury).—The posterior tibial and dorsalis pedis pulses were normal at the ankle. There was moderate clawing of the toes with slight contracture in long extensor and flexor tendons.

Motor: There was wasting and paralysis of all the intrinsic muscles of the foot—extensor digitorum brevis as well as those of the sole—and all these muscles gave R.D. on electrical testing. All the muscles of the leg showed full voluntary power and a normal response to faradism.

Sensory: There was loss of sensation to light touch and pin-prick in the areas of distribution of all the deep nerves of the leg—anterior tibial, posterior tibial, and musculo-cutaneous—and in the area of the sural nerve (*Fig.* 334). Deep pressure sense and joint sense were absent in the distal part of the foot.

Sweating: There was anhydrosis in the anæsthetic area.

RECOVERY.—The first sign of sensory recovery was noted 6½ months after injury and it slowly progressed in all areas in a manner indicative of nerve regeneration. Seventeen months after injury recovery of sensation was almost complete in musculocutaneous, anterial tibial, and sural areas, but there was still some loss to light touch in the distal part of the sole. Some return of voluntary power and some response to faradism were noted in abductor hallucis. During recovery there was a very marked 'protopathic' type of response to pin-prick, especially on the sole. There was a definite delay (about 1·5 seconds) in the appreciation of the stimulus and the sensation evoked thereby was of an extremely unpleasant character. Rubbing the skin of the sole also gave rise to a very unpleasant sensation ('plantar dysthesia'), and at this stage of recovery there was a good deal of pain on walking. These phenomena became much reduced in intensity as sensation to light touch was regained. The slight shortening of the long flexor and extensor tendons gradually developed into a fairly severe contracture which has recently been treated by operation.

Case No. 2.—T. G., aged 25.

HISTORY.—This soldier was struck by a car and sustained a 'bumper' fracture of the upper end of the tibia. Despite considerable swelling of the leg a closed plaster was applied, but had to be removed the following day because of poor circulation in the foot. Skeletal traction on a Braun's splint was substituted and later a drop-foot was noticed. The patient could give no clear history regarding the onset of anæsthesia.

EXAMINATION.—The findings were similar to those in *Case* 1, except that extensor hallucis longus was paralysed and there was marked weakness of other muscles supplied by the lateral popliteal nerve. Sensation was present in the sural distribution, but lost in the distal part of the saphenous area (*Fig.* 335).

RECOVERY.—The first sign of sensory recovery was noted seven months after injury, and later delayed 'protopathic' response to pin-prick was much in

evidence. When last examined fifteen months after injury all muscles were active, though the anterior tibial muscles were still weak. Sensory loss was

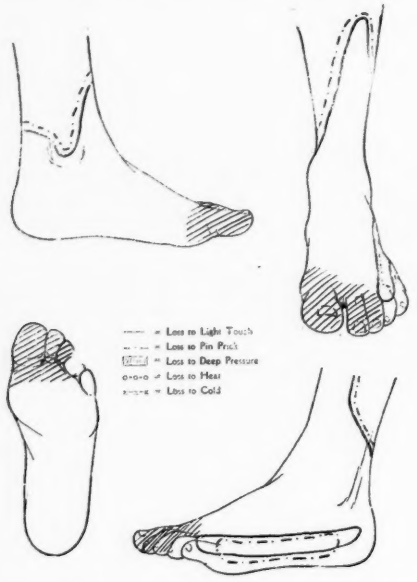

Fig. 335.—The sensory loss involves the distal part of the saphenous distribution, but the sural area has escaped.

confined to the distal part of the sole and the delayed response to pin-prick could not be demonstrated. There was no obvious muscle contracture.

Case 3.—R. L., aged 18.

HISTORY.—Both legs crushed by a heavy boulder during an air raid. The right leg had to be amputated. On the left side there was no fracture and the skin was intact, but the leg was greatly swollen for about a week after the injury. It was not until seventeen months after the injury that this patient was first seen at the Centre.

EXAMINATION.—There was paralysis and R.D. in all intrinsic muscles of the foot and loss of sensation to light touch over the whole foot except in the saphenous distribution and over a small area on the sole. There was a corresponding loss to pin-prick, except that over most of the sole there was hyperalgesia with a delayed response (*Fig.* 336).

RECOVERY.—Thirty months after injury there was practically complete recovery of sensation and some return of voluntary power to the intrinsic muscles of the sole. There was no contracture.

Case 4.—R. McN., aged 19.

HISTORY.—Closed comminuted fracture of the tibia at the junction of its middle and lower thirds in a railway accident.

EXAMINATION.—There was loss of function in all three deep nerves of the leg and in the sural nerve. The long extensor and long flexor of the great toe were paralysed, as well as the intrinsic muscles of the foot.

RECOVERY.—Nine months after injury there was some recovery in the posterior tibial nerve.

Case 5.—D. B., aged 37.

HISTORY.—Closed comminuted fracture of tibia and fibula caused by a heavy weight falling on the leg. The fracture was about the middle of the leg.

EXAMINATION.—The findings were very similar to those in *Case* 4, except that extensor hallucis longus was the only leg muscle affected.

RECOVERY.—Fourteen months after injury there was considerable sensory recovery in all affected nerves and some voluntary power in abductor hallucis.

Case 6.—J. H., aged 20.

HISTORY.—Accidental through-and-through bullet wound just below the knee, which caused a comminuted fracture of the upper end of the tibia without displacement. The leg was put up in plaster. Two months after injury the patient was examined at another Peripheral Nerve Injuries Unit, where all the muscles below the knee were found to be paralysed and to give R.D. There was sensory loss in the areas of distribution of all the deep and superficial nerves below the knee. The posterior tibial pulse was palpable at the ankle. *Exploration*: The nerves and vessels were explored in the popliteal fossa and upper part of the leg, but no abnormality was seen.

RECOVERY.—Three months after injury there was some return of voluntary power to the calf muscles and peronei. Twenty-one months after injury voluntary power had returned to all the muscles of the leg and sensory recovery was progressing, but a severe contracture had developed in both anterior and posterior tibial muscles.

EXPLORATION.—To correct the deformity resulting from the above contractures an operation was performed by Mr. Roland Barnes, and this provided an opportunity of inspecting the structures in the lower part of the posterior compartment of the leg. Flexor hallucis longus showed the typical appearance of a true Volkmann's ischæmia, the muscle substance

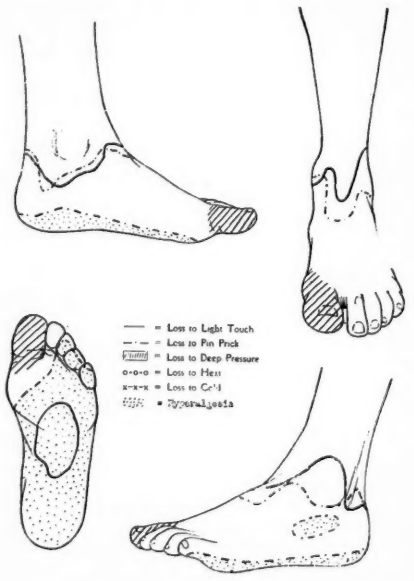

Fig. 336.—In this case there has been some recovery of sensation in the posterior tibial and sural areas.

being replaced by a homogeneous yellow mass with a sheath of dense fibrous tissue. All the other muscles appear normal. There was good pulsation in the posterior tibial artery, but none in the peroneal.

BIOPSY.—Specimens were obtained from flexor hallucis longus, flexor digitorum longus, and soleus. On histological examination only the first-named

showed the typical mass necrosis and fibrous tissue replacement of Volkmann's ischæmic contracture (*Fig.* 337). The others showed only some degeneration of muscle-fibres with interfibrillary fibrosis on the surface of the muscles.

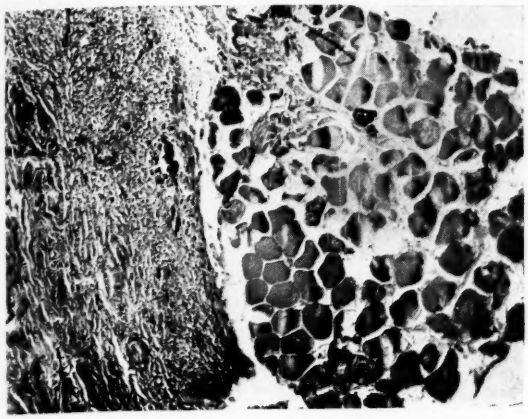

FIG. 337.—Transverse section of flexor hallucis longus eighteen months after injury, showing completely necrotic muscle with dense scar tissue at its periphery.

Case 7.—J. M., aged 25.

HISTORY.—Through-and-through bullet wound of the leg about its middle, which caused a comminuted fracture of the fibula. The wounds were excised and sutured. Three days later the leg became very swollen and tense, and anæsthesia of the foot was noticed.

EXAMINATION.—Three weeks after injury the posterior tibial and dorsalis pedis pulses were normal at the ankle. There was loss of function in all three deep nerves of the leg with paralysis of the long flexor and long extensor of the great toe.

EXPLORATION.—Some months later the posterior tibial nerve was explored. There was no sign of division, but the nerve was thin and atrophic below the level of the injury.

RECOVERY.—Twenty-one months after injury there was no motor recovery, but considerable recovery of sensation, especially in the musculo-cutaneous distribution.

Case 8.—D. McK., aged 33.

HISTORY.—Through-and-through bullet wound of the thigh. The track of the bullet did not pass near the sciatic nerve, but it did traverse Hunter's canal.

Two days later there was a marked bruit and visible pulsation in the region of the femoral vessels, but the pulse was palpable at the ankle. There was no evidence of nerve lesion at this time. During the next six weeks the patient was in considerable pain and the pulsating swelling in the thigh gradually enlarged.

44th day: Notes state that there were no active movements at the knee, ankle, or toes, and no sensation to touch in the foot or to passive movements of the toes.

46th day: *Operation.* A communication between the superficial femoral artery and vein was excised and a large hæmatoma evacuated from the thigh. The wound was closed without drainage. The day following this operation the foot was very cold, but thereafter the circulation rapidly improved.

51st day: There were slight flickering movements in the toes and, though sensation was normal above the ankle, it was lost over the foot except on the medial side. Joint sense was absent in the toes. There was a moderate degree of swelling of the thigh.

52nd day: Active movements at the ankle and toes were present, and sensation was present over the greater part of the sole as well as along the medial border of the foot.

53rd day: Marked deterioration in movements at the ankle and toes and no sensation to touch below the knee.

55th day: Anæsthesia now more or less 'stocking' below the tibial tuberosity.

58th day: Only slight flickering movements at ankle and toes. Circulation through the foot good, despite some œdema.

60th day: *Forty-one ounces* of thick altered blood and pus evacuated from thigh and cavity drained.

71st day: Movements slowly returning to leg and foot.

98th day: *Electrical Reactions*: No response to faradism in muscles of leg.

Seven and a half months after injury there was good voluntary power in the calf muscles and weak voluntary activity in some of the proximal muscles of the leg. Anæsthesia still present.

EXAMINATION.—When first examined at the Centre twelve months after injury, the pulse at the ankle was strong and there were no signs of any muscle contracture.

Motor: The muscles of the thigh and the more proximal muscles of the leg had good voluntary power, but the long extensor and long flexor of the great toe were very weak and all the intrinsic muscles of the foot were paralysed.

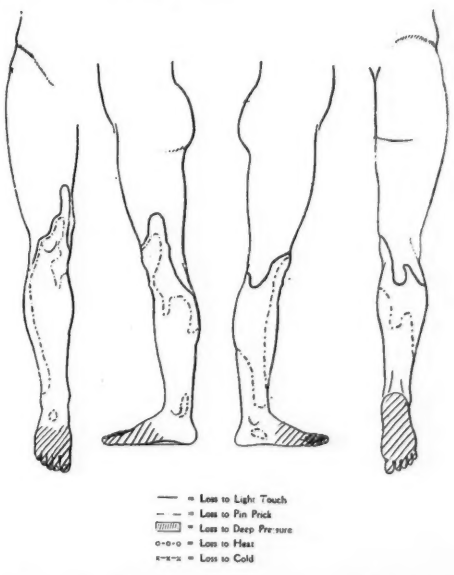

FIG. 338.—Showing more or less stocking anæsthesia to light touch, but with areas of sensitivity to pin-prick in the proximal parts of the areas of distribution of saphenous, musculocutaneous, posterior tibial, and sural nerves.

Sensory: Sensation to light touch was lost over the whole sciatic area and also in the areas supplied by the saphenous nerve and the medial cutaneous nerve of the thigh. There was slight sensation to pin-prick in the proximal part of the areas supplied

by saphenous, musculo-cutaneous, sural, and posterior tibial nerves (*Fig.* 338). Deep pressure and joint sense were lost over the distal half of the foot.

Vasomotor: The affected foot was warmer than the other.

Sudomotor: Small patches of sweating were present below the knee only in those areas noted above where there was some sensation to pin-prick.

RECOVERY.—When re-examined fifteen months after injury there were no obvious signs of further recovery in the nerves, and a slight contracture had developed in the calf and anterior tibial muscles.

Eighteen months after injury there was some return of sensation to pin-prick in the posterior tibial and sural areas in the foot, with corresponding areas of sweating.

Case 9.—C. P., aged 34.

HISTORY.—This patient underwent a high ligation operation for bilateral varicose veins. On the right

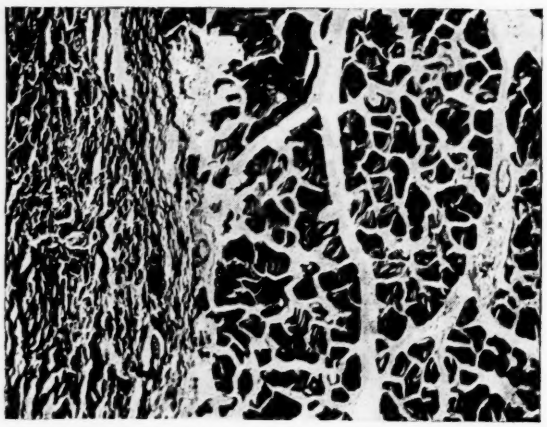

FIG. 339.—Section of tibialis anterior, showing mass necrosis of muscle with scar tissue at the periphery. There is remarkably little cellular activity.

side the operation was difficult owing to the presence of a mass of dilated veins in the neighbourhood, and after 10 c.c. of sodium morrhuate had been injected into the distal segment the ligature slipped and there was brisk hæmorrhage into the wound from this portion of the vein. The vein had to be exposed at a lower level and ligated here before the bleeding was controlled.

The day following the operation the patient complained of very severe pain in the calf muscles and in the popliteal region. The gastrocnemius was swollen and very tender, and there was pain on pressure over the popliteal space. The leg and foot were pale, with dilated superficial veins. In addition the peroneal muscles were found to be paralysed, and there was loss of sensation over the whole foot except along its medial border.

The severe pain continued and two weeks after operation there was paralysis of peronei and tibialis anterior and marked weakness of all other muscles below the knee. There was complete sciatic sensory loss. Three weeks after operation there was a further brisk hæmorrhage from the wound, necessitating re-opening under general anæsthesia.

Three months after operation the patient was admitted to a Peripheral Nerve Injuries Unit where, apart from weak voluntary power in the muscles of the posterior compartment of the leg, there was found to be a complete sciatic palsy. Subsequent notes from this Unit recorded progressive recovery in the medial popliteal nerve with a marked delayed response to pin-prick at one stage and little recovery in joint sense. The lateral popliteal nerve showed practically no sign of recovery.

EXAMINATION.—When first examined some months later no pulse could be felt at the ankle, and the popliteal pulse was weak compared with the opposite side. The foot became pale when elevated and cyanosed when dependent. There was some contracture of anterior tibial and peroneal muscles, but not in the posterior tibial group. The muscles in the anterior compartment felt extremely indurated.

Motor: There was satisfactory voluntary power in all the muscles of the posterior compartment, but apart from a flicker in the proximal fibres of tibialis anterior there was no activity in the muscles of the anterior and lateral compartments of the leg or in the intrinsic muscles of the foot.

Sensory: Complete sensory loss in the distribution of the lateral popliteal nerve, but considerable recovery in the medial popliteal, with some residual delayed response to pin-prick on the plantar aspect of the toes.

BIOPSY.—A piece of tibialis anterior was removed for examination. The naked-eye appearance of the muscle was typical of Volkmann's ischæmia and histological examination confirmed the presence of this condition (*Fig.* 339).

RECOVERY.—Three months after the above examination there was still further sensory recovery, especially in the medial popliteal distribution.

UPPER LIMB

Case 10.—A. B., aged 28.

HISTORY.—Whilst cleaning his rifle this soldier accidentally shot himself through the right axilla. Profuse hæmorrhage from the wounds necessitated repeated blood transfusions but the axilla was not explored.

EXAMINATION.—On admission to the Centre ten days after injury the whole limb was grossly swollen but the radial pulse was palpable at the wrist.

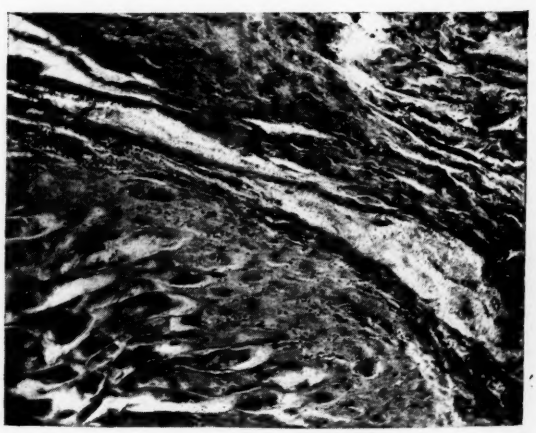

FIG. 340.—Longitudinal section of biceps brachii in a case of injury to the axillary artery. The muscle is necrotic and is being replaced by dense scar tissue. Note cellular activity at the junction of muscle and fibrous tissue.

Motor: There was paralysis of all muscles below the shoulder with the exception of deltoid and triceps.

Sensory: Sensory loss extended up to the level of the middle of the arm anteriorly and to the middle of the forearm posteriorly.

After the swelling had been reduced by placing the limb on an abduction splint it was noticed that the biceps was extremely indurated; it felt, in fact, like a typical Volkmann's ischæmia, and this diagnosis was later confirmed by biopsy (*Fig.* 340). No pulsation could be detected in the brachial artery, but the profunda vessel was pulsating strongly.

RECOVERY.—Ten months after injury there was quite good voluntary power in all muscles receiving their nerve-supply above mid-forearm, but no recovery in the more distal muscles. Sensation had recovered as far as the wrist, except in the superficial radial distribution where recovery was complete.

Fifteen months after injury there was some recovery of sensation to pin-prick in the median distribution.

Case 11.—J. C., aged 28.

HISTORY.—Through-and-through bullet wound of the axilla. The axilla was explored a few hours after injury and a large axillary hæmatoma was evacuated. The axillary artery was found to have been damaged and the radial nerve had been divided. The median and ulnar nerves appeared to be intact. The artery was ligated.

EXAMINATION.—

Motor: Five months after injury there was, in addition to a complete radial-nerve palsy, paralysis of all the intrinsic muscles of the hand. All the paralysed muscles gave R.D.

Sensory: Complete sensory loss in median, ulnar, and superficial radial areas.

A hard mass of almost cartilaginous consistence was felt in the substance of the long and lateral heads of triceps, and the muscles of the forearm also felt somewhat indurated. Subsequent biopsy of these muscles showed the typical Volkmann appearance only in triceps (*Fig.* 341).

EXPLORATION.—Division of the radial nerve was confirmed and suture was performed. There was no obvious abnormality of the median and ulnar nerves.

RECOVERY.—Sensory recovery is taking place in the median area, but there is not as yet any activity in the intrinsic muscles of the hand.

Case 12.—G. E., aged 28.

HISTORY.—G.S.W. of upper arm, causing a compound comminuted fracture of the humerus. The wounds were excised and plaster was applied. Following this the circulation in the hand was very poor and the forearm and hand became cyanosed. The radial pulse was absent for the first few days after injury.

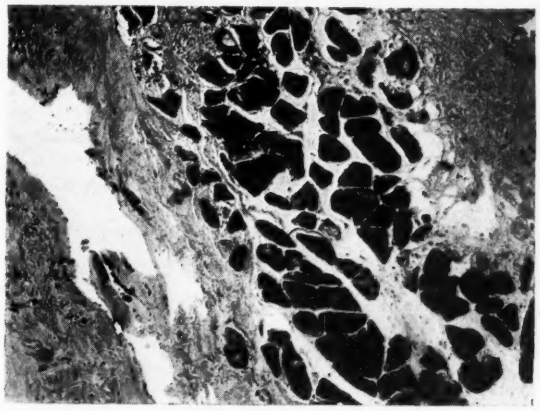

FIG. 341.—Section of triceps six months after injury to axillary artery. Note dead muscle mass being replaced by fibrous tissue.

EXAMINATION.—The findings were almost the same as those in the previous case, including division of the radial nerve. Volkmann's ischæmic contracture of the forearm muscles was suspected, but muscle biopsy did not support this.

Case 13.—T. P., aged 32.

HISTORY.—Closed fracture of the lower end of the ulna as a result of a motor-cycle accident. A skin-tight plaster was applied, and when this was removed some weeks later there were pressure sores in the region of the wrist—one of them immediately over the radial artery—and there was loss of sensation in the fingers.

EXAMINATION.—Five months after injury the radial pulse was present even beyond the site of the pressure sore. There was paralysis and R.D. in all the intrinsic muscles of the hand, with very poor responses to galvanism. Sensation to light touch was absent in all the fingers, but that to pin-prick was lost only over the distal phalanx of the index.

At first no contracture of muscles was noticed, as full flexion and extension of the fingers was possible.

FIG. 342.—Contracture of interossei does not prevent flexion of interphalangeal joints when metacarpophalangeal joints are flexed.

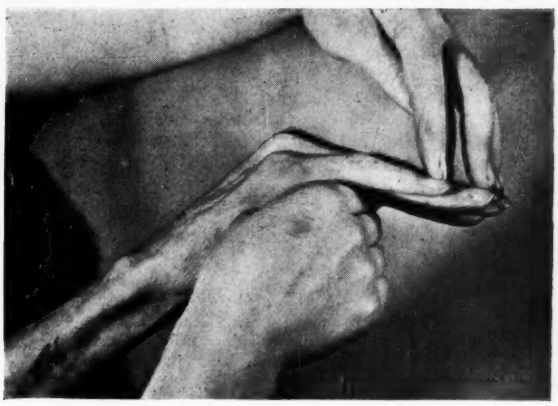

FIG. 343.—Contracture of interossei prevents passive flexion of interphalangeal joints when metacarpophalangeal joints are held moderately extended.

More careful examination, however, revealed the presence of a moderately severe contracture of the interossei because of which it was found impossible passively to extend the metacarpophalangeal and flex the interphalangeal joints at the same time (*Figs.* 343, 343). There was also some restriction of lateral movements at the metacarpophalangeal joints.

RECOVERY.—Eight months after injury voluntary power had returned to most of the intrinsic muscles of the hand and sensation had almost completely recovered. The contracture of the interossei was becoming more severe.

OPERATION.—The tendons of the contracted interossei were divided by a palmar approach. This resulted in a considerable improvement in the function

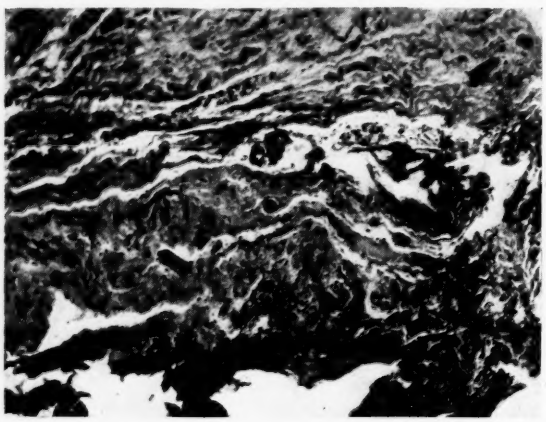

FIG. 344.—Section of fourth dorsal interosseous muscle. Note dense fibrous tissue engulfing a few dead muscle-fibres.

of the hand. At operation it was noted that the lumbrical muscles looked absolutely healthy. Portions of the affected interossei were removed for histological examination and the sections were most suggestive of Volkmann's contracture (*Fig.* 344).

Case 14.—B. T., aged 25.

HISTORY.—Wrist crushed between two vehicles and sustained a fracture-dislocation. Shortly after the injury the wrist became very swollen and the patient began to experience tingling in the middle three digits. The tingling persisted for four or five days and then the fingers became numb.

EXAMINATION.—

Motor: Voluntary power was normal in all intrinsic muscles of the hand.

Sensory: There was anæsthesia to light touch over the whole of the hand, with the exception of the area supplied by the dorsal branch of the ulnar nerve. Sensation to pin-prick absent over the distal half of the lateral three digits. Joint sense absent in the interphalangeal joints of the lateral three digits.

Case 15.—Mrs. M. S., aged 61.

HISTORY.—Fracture-dislocation of the right shoulder. Notes of examination before reduction state that there was paralysis of the circumflex nerve, but no sign of involvement of median, ulnar, or radial nerves. The dislocation was reduced and the following day there was marked swelling of the upper arm and 'impairment' of sensation in the hand, with inability to extend the interphalangeal joints. Three days after injury it was noticed that there was no pulsation in the arteries of the limb below the subclavian, though the hand was warm and showed no evidence of impairment of the circulation.

EXAMINATION.—When admitted to the Centre one week after injury there was still considerable swelling and bruising of the shoulder region and upper arm. No pulsation could be felt in the arteries below the subclavian.

Motor: In addition to paralysis of the deltoid, no activity could be detected in any of the intrinsic muscles of the hand or in the muscles supplied by the ulnar nerve in the forearm.

Sensory: Ulnar, complete sensory loss. Median, loss of sensation to light touch and almost complete loss of sensation to pin-prick. Superficial radial, loss of sensation to light touch, but very striking delayed response to pin-prick.

RECOVERY.—Two days after the first examination the following points were noted:—

Motor: Weak voluntary power had returned to flexor carpi ulnaris.

Sensory: Ulnar, complete sensory loss. Median, loss of sensation to light touch persisted but a delayed response to pin-prick was present throughout the median area. Superficial radial, there was some appreciation of light touch and an immediate response to pin-prick.

Joint sense was absent in all the interphalangeal joints, except in the thumb.

Two days later still the results of sensory testing showed: Ulnar, striking delayed response to pin-prick and firm pressure. Median, as before.

Some weeks later the condition had remained unchanged, with no return of light touch sensation in ulnar or median areas and paralysis of all intrinsic muscles of the hand. The 'slow' pain response persisted.

SUMMARY OF CLINICAL FEATURES

1. A history of injury to the limb followed by gross swelling or other signs of severe circulatory disturbance.

2. Evidence of disturbance of function, generally amounting to complete paralysis, in all the deep nerve-trunks of the affected part.

3. Sensory loss is generally more obvious than motor paralysis and, unless the nerves are 'concussed' by the initial injury, the anæsthesia is of gradual onset, commencing in the digits and spreading centripetally. It is frequently of the 'stocking' or 'glove' type and is sometimes thought to be 'hysterical'.

4. There may be some dissociation between various forms of sensation, especially during the early stages and during recovery, e.g., light touch sensation and joint sense are more likely to be lost than is sensation to pin-prick. A delayed response to pin-prick is a notable feature in most cases at some stage.

5. Motor loss, when present, is frequently confined to the intrinsic muscles of the hand or foot, though it may affect some of the more proximal muscles.

6. Muscle contractures are frequently present in the later stages, but they are usually of mild degree and are seldom of the true Volkmann type.

7. Spontaneous recovery occurs in most cases but, apart from that which may occur during the first few weeks after injury, it is very slow and is dependent upon nerve regeneration.

AETIOLOGY

In considering the aetiology of the multiple nerve lesions in these cases the following possibilities suggest themselves :—

1. Direct mechanical injury to the nerve-trunks.
2. Involvement of nerve-trunks in scar tissue or callus.
3. Peripheral nerve-endings affected by circulatory disturbance.
4. Traumatic arterial spasm (or arterial ligation).
5. Pressure beneath the deep fascia due to extravasation of blood and tissue fluids, causing ischæmia of the nerve-trunks.

These factors will be considered in the above order. Hysteria was considered as a possible cause, but was ruled out because of the presence of muscular wasting and R.D. in paralysed muscles and loss of sweating in anæsthetic areas.

1. Direct Mechanical Injury to the Nerve-trunks

The multiplicity of the nerve lesions is obviously a point against this as a possible cause unless the lesions can be regarded as being due to 'concussion' of the nerve-trunks. If that were so early recovery would be expected. Moreover, in those cases where the nerves were explored (*Cases* 6, 7, and 11) there was no evidence of division or of direct pressure on the nerves, with the exception of the radial nerve in *Cases* 11 and 12. The cases with a history of gradual onset also preclude such a cause.

2. Involvement of Nerves in Scar Tissue or Callus

This could possibly be a cause in only a few of the cases, and even in those there are obvious objections. In nearly every case the nerve lesions were obviously present long before such factors could have become operative.

3. An Effect of the Circulatory Disturbance on the Peripheral Nerve-endings

That this may be a cause is suggested by the fact that the neurological defects are most marked in the distal parts of the limb. One would naturally expect that when the blood-supply to a limb is reduced to the point of ischæmia the effect on the nerves would be most intense at the periphery. It has, however, been shown experimentally by Lewis and his co-workers (1931) that although the *effects* are first observed at the periphery, they are due to ischæmia of the nerve-trunks. This view has received the support of other workers (Gasser, 1934; Clark, Hughes, and Gasser, 1935; Thompson and Kimball, 1936). Moreover, in the present series of cases, careful charting of the sensory loss and the manner of its recovery is definitely in favour of the lesion being situated in the nerve-trunks near the site of injury.

4. Traumatic Arterial Spasm (or Arterial Ligation)

This is a subject about which much has been written in recent years (Griffiths, 1940; Cohen 1940–41; Barnes and Trueta, 1942). The condition is now generally recognized to be a cause of Volkmann's ischæmic contracture and, in view of the frequent association of peripheral nerve lesions with the latter condition, it must be considered to be a likely aetiological factor in some of the cases under discussion.

The question can hardly be discussed without reference to Volkmann's ischæmic contracture.

Some Observations on Volkmann's Contracture.—There has been considerable renewal of interest in this condition in recent years, especially since the publication of Griffith's Hunterian Lecture on the subject (1940). For some years prior to this the venous obstruction theory of its aetiology was generally held, but now the tendency is to regard the condition as being due to arterial injury and spasm. Recently Albert and Mitchell (1943) put forward the view that the Volkmann syndrome can be caused either by temporary arterial obstruction or by venous occlusion. On the whole the evidence seems to be in favour of Griffith's contention that the true Volkmann's ischæmia with massive necrosis of muscle-fibres and their subsequent replacement by fibrous tissue is due to deprivation of arterial blood-supply generally as a result of traumatic arterial spasm. Nevertheless there is little doubt that a form of muscle contracture clinically resembling the true Volkmann type, though probably without the typical histological picture, can occur from venous or capillary occlusion.

The condition occurs most frequently in the flexor muscles of the forearm, usually following supracondylar fracture of the humerus. It is perhaps unfortunate that attention has been so focused on this region that it is probable the condition has frequently passed unrecognized in other muscle groups. Yet there is no reason why, given the necessary aetiological conditions, it should not be found elsewhere, and, in fact, a number of cases have been described.

It is perhaps not without interest that of the 15 cases of nerve lesions described above, 5 show fairly definite evidence of Volkmann's necrosis, and in none of these is it in the forearm. The distribution is as follows: Flexor hallucis longus (*Case* 6), Anterior tibial group (*Case* 9), Biceps brachii (*Case* 10), Triceps (*Case* 11), and Interossei of the hand (*Case* 13). In all these five cases there was direct or indirect evidence of arterial injury, and the question arises as to what determines the site of the subsequent muscle necrosis. The effect of a main arterial lesion on the limb depends largely on the condition of the collateral circulation. To quote Griffiths (1940): "If this be free, all is well. If it be blocked completely, gangrene results. If blockage be incomplete, but too severe or too prolonged for

complete recovery of the limb, then necrosis will occur in the tissues whose vascular demands are highest." There would seem to be three main factors causing obstruction of the collateral circulation—spasm of collaterals by local sympathetic reflexes, thrombosis, and obstruction by swelling, with or without external constriction—and these, as well as the situation of the arterial lesion will determine which muscles will be affected. In the most usual type of true Volkmann's contracture affecting the forearm flexors, the more distal muscles of the limb, e.g., the intrinsic muscles of the hand, are not necessarily affected. It may be that these muscles are more resistant to ischæmia owing to their small size, but it is more likely that the occurrence of necrosis in the forearm flexor group is determined by extension of the arterial spasm (or thrombosis) to involve the actual muscular branches of the vessel, thus isolating the muscles which they supply from any collateral circulation which may be present in the limb and causing massive necrosis. If this be so it would afford a satisfactory explanation of the confinement of necrosis to the individual muscles or groups of muscles in the five cases in our present series.

Effect of Arterial Lesions on Nerves.—Though nerve lesions are well known to be frequently associated with Volkmann's ischæmic contracture, they have generally been considered to be due to coincident direct injury to the nerves or to subsequent involvement of the nerve-trunks in the fibrosis which inevitably follows and is, in fact, part of this condition. It seems, however, that many reported cases of Volkmann's contracture have had no nerve lesions at all, so that it would seem to be possible for arterial injury and associated spasm to cause necrosis of muscle without at the same time rendering the nerve-trunks sufficiently ischæmic to cause peripheral degeneration or even temporarily to abolish conduction in them.

It is well known that the principle artery of a limb can be ligated without, in most cases, causing any permanent damage to either muscle or nerve. If, however, the circulation to the limb is completely arrested, as by a tourniquet at its root, conduction in the nerves distal to the tourniquet begins to fail within half an hour (Bentley and Schlapp, 1943, a). Even after conduction has been abolished in the nerves of the ischæmic limb the muscles will still respond to direct stimulation. That is, in ischæmia the *function* of the nerves fails before that of the muscles. But, according to Lewis (1936), long-continued ischæmia kills muscle more readily than it kills nerve. The actual times are not known, but Lewis suggests that muscles are killed after six to eight hours of ischæmia while nerves will recover after twelve to twenty-four hours.

This question of the relative susceptibilities of muscle and nerve is obviously important in our present discussion, for if, as seems likely, muscle is more susceptible than nerve, we should not expect to find degeneration of nerve in a limb which does not also show necrosis in its muscles. The recent work of Bentley and Schlapp (1943, a) led them to conclude that a small but definite blood-supply is essential for the maintenance of *conduction* in nerve-trunks, while Bulbring and Burn (1939) maintained that conduction can fail when the blood-supply is reduced below a certain level even though the ischæmia of the nerve is not complete. What does *not* seem to have been established is the degree of ischæmia and the length of time it must act before peripheral degeneration of the nerve becomes inevitable.

In an exhaustive review of the anatomical evidence relating to the blood-supply of nerves, Adams (1942) summarizes by saying that this evidence " shows beyond doubt that all nerves receive a blood-supply and that it is derived from regional vessels which contribute, however, to the formation of a longitudinal vascular pathway along the nerve ". Writing on the subject of the innervation of these vasa nervorum, he states that the larger branches of the nutrient arteries running in the epineurium and perineurium have a well-developed tunica media composed of smooth muscle-fibres and that non-medullated nerve-fibres (nervi nervorum) have been demonstrated in the same situations. Though the significance of these nerves is not known for certain, Sappey (1889) regarded them as being vasomotor in function. If this be so it suggests a possible explanation of the nerve lesions which sometimes occur in cases of traumatic arterial spasm, for the spasm may involve the regional vasa nervorum in the same way as it may involve the muscular branches of the artery. Whether such a mechanism would cause a sufficient degree of ischæmia of the nerve-trunks to produce peripheral degeneration is doubtful unless there be some additional interference with the collateral supply via the longitudinal pathway. Such additional interferenee might well be supplied by swelling of the limb.

To summarize, the evidence at present available suggests that on the whole it is unlikely that traumatic arterial spasm can *by itself* cause ischæmic degeneration of nerve-trunks without at the same time producing necrosis in the muscles. The only way in which it could do so—and this is purely hypothetical—would be by extension of the local arterial spasm to involve the regional vasa nervorum.

5. Effect on the Nerve-trunks of Pressure beneath the Deep Fascia

The work of Lewis, Pickering, and Rothschild (1931) is of particular interest in this connexion. During the course of a large number of most interesting experiments on nerve ischæmia in the intact human subject, these workers designed a special clamp by means of which the nerve-trunks of the arm could be compressed without greatly affecting the circulation in the rest of the

limb. Using this clamp they found that pressures as low as 60–70 mm. Hg were sufficient to block conduction in the nerve-trunks, and they showed that the block was due to ischæmia. The onset of the anæsthesia so produced was centripetal in type; it commenced about sixteen minutes after the application of the clamp in the tips of the fingers and gradually spread proximally until it eventually involved the whole area distal to the clamp. Moreover, there was some dissociation of sensation; for instance, loss of tactile and joint sense occurred earlier than loss of pain appreciation, and the relative susceptibility of the various types of fibres subserving different forms of sensation was more or less dependent upon their size. The large diameter fast-conducting fibres (A fibres of Gasser) were more susceptible than the small diameter slow-conducting fibres (C fibres of Gasser). About the time the whole hand had become anæsthetic voluntary power was lost in all the intrinsic muscles of the hand, and later paralysis of the forearm muscles followed.

In these experiments the pressure was never maintained for more than an hour and when it was released there was rapid restoration of function in the nerves.

In most of the cases at present under discussion the history of gross swelling of the limb, the mode of onset of anæsthesia, and the fact that those nerves lying outside the deep fascia at the level of the swelling often escaped, suggest that pressure of extravasated blood and fluid exudate beneath the rather unyielding fascial envelope might reproduce much the same conditions as were operative in Lewis's clamp experiment. The actual pressure which can develop beneath the deep fascia in such cases is, so far as I am aware, not known, but 60–70 mm. Hg would seem to be a possibility.

Recently Bentley and Schlapp (1943, b) have investigated the effects of direct pressure applied to a nerve-trunk in the hind limb of the cat. They found that a pressure of 60 mm. Hg was sufficient to render ischæmic a 4-cm. length of nerve-trunk, but that in this case nerve-block was not produced because of diffusion of oxygen from the adjacent vascularized nerve. In view of this it might well be argued that in the present series of cases there would surely be sufficient oxygen available by diffusion from the compressing fluids to maintain conduction in the nerves. One can only conjecture that the combination of the pressure beneath the deep fascia, *plus* the general anoxia of the limb, *plus* possible thrombosis in the veins and capillaries of the nerves—all involving a considerable length of the trunks—is sufficient to abolish conduction. And if such conditions be allowed to persist for a sufficient length of time, probably about 24 hours, it is likely that peripheral degeneration of the nerves would occur.

This 'pressure ischæmia' theory of causation seems most adequately to explain the clinical findings in most of the cases under consideration. The mode of onset of anæsthesia, the nerve territories involved, the distribution of muscle paralysis, and the dissociation of sensation sometimes observed are all in accordance with the observations of Lewis et al. (1931). It will be noted that in the lower limb cases the long muscles of the hallux are frequently the only leg muscles affected. They receive their nerve-supply at a lower level than the others. The question of dissociation of sensation was subsequently re-investigated by Lewis and Pochin (1937) and has also been fully dealt with by Wortis, Stein, and Jolliffe (1942). The markedly delayed response to pin-prick which was such a noteworthy feature of many of the cases is of special interest. When observed within a few days of injury (as in *Case* 15) it provides a convincing demonstration of the fact that the slow-conducting 'second' pain fibres are relatively resistant to ischæmia. The phenomenon is, however, more frequently observed shortly after sensory recovery first becomes evident during regeneration. It is sometimes observed during the early stages of recovery after nerve suture, but is much more striking following ischæmia. This may be due to fine and poorly myelinated nerve-fibres growing through the ischæmic region of the nerve-trunk more readily than the fibres of larger diameter, or possibly to delayed re-myelinization of regenerated fibres. Certainly it is found that the phenomenon disappears as sensation to light touch is regained.

This 'pressure ischæmia' theory is not put forward without the realization that there are certain physical problems involved which cannot be adequately dealt with in this paper, e.g., the question of pressure gradients within tissues of such different consistency as nerve and muscle and the effect of this on the relative degrees of ischæmia in each.

However, the most convincing evidence in favour of the theory is provided by two cases, notes on which have kindly been supplied to me by Professor Seddon, of Oxford:—

Case A.—Admitted to hospital complaining of weakness and loss of sensation in the right hand.

PREVIOUS HISTORY.—Typical history of hæmophilia, the patient having had at various times hæmophilic arthritis of both knees, both ankles, and right elbow; also melæna and severe bleeding after tooth extraction.

HISTORY.—Three months prior to admission the patient fell and struck his elbow against a sideboard. Fourteen hours later the soft tissues of the upper forearm began to swell and show bruising. The swelling rapidly increased and for three weeks the forearm and hand were so swollen that little movement was possible. When the swelling subsided the patient noticed weakness and loss of sensation over the whole hand. This had persisted up to the time of admission, except for some return of sensation in the superficial radial distribution.

EXAMINATION.—There was a typical claw-hand deformity of combined median and ulnar nerve palsy. Wasting, paralysis, and R.D. were present

in all intrinsic muscles of the hand, while all the forearm muscles were normal. There was typical sensory loss in ulnar and median areas, except for some sensation to pin-prick on the radial half of the palm. In the superficial radial distribution sensation was diminished but not lost.

Conclusion: "Combined median and ulnar palsy with transient lesion of radial. Level of lesion probably lower forearm and probably of the compression type. No evidence of Volkmann's ischæmic contracture."

RECOVERY.—Six months after injury there was no change in motor functions, but considerable recovery of sensation to pin-prick in the median area.

Eight months after injury power had returned to the thenar muscles and sensation to pin-prick had recovered in the ulnar area. "Character of pain: Threshold slightly increased. Appreciation slightly delayed."

Twelve months after injury there was practically full power in median intrinsic muscles, but the ulnar intrinsics, though acting, had lagged behind. Sensation had practically recovered, except for residual loss to light touch, in the distal part of the ulnar distribution.

The function of the nerves continued to improve, but even two years after injury there was still some weakness of ulnar intrinsics and some imperfection of the more specialized sensory functions (stereognosis and 2-point discrimination) in the ulnar area.

Case B.—Admitted to hospital complaining of weakness and loss of sensation in the left hand.

PREVIOUS HISTORY.—Typical history of hæmophilia since early childhood.

HISTORY.—Six weeks prior to admission a ladder fell across the left forearm. Twelve hours later the hand and forearm became very swollen, and the hand became numb and useless. When examined later the following points were noted: Radial pulse present; muscles of forearm soft and showing active contraction; paralysis of all intrinsics of hand; sensory loss of the glove type extending proximally as far as the level of the radial styloid.

EXAMINATION.—The findings were almost identical with those in the previous case, except that there was complete anæsthesia in the superficial radial distribution.

RECOVERY.—Some recovery of sensation was first noticed seven months after injury and slowly progressed in all three nerves.

Eighteen months after the above injury the patient suffered further slight damage to the forearm with recurrence of similar but less severe symptoms of nerve injury.

The main points of interest in these two cases are:—

1. The trivial nature of the injury, which could not conceivably have been directly responsible for the multiple nerve lesions.

2. The lack of any evidence of damage to main arteries.

3. The relation of the development of gross swelling to the onset of loss of function in the nerves.

4. The involvement of three nerve-trunks all lying beneath the deep fascia at the level of the injury while the superficial nerves were unaffected.

5. The manner in which recovery took place indicates that the lesion was situated in the nerve-trunks and was not a primary effect on the peripheral nerve-endings.

A case in which an ilio-psoas hæmatoma affected the femoral nerve has also been described by Seddon in a paper in which he reviews the association of hæmophilia and lesions in the nervous system (Seddon, 1930).

SUMMARY OF AETIOLOGY

While most of the nerve lesions described in the present series of cases can be most satisfactorily explained by ischæmia of the nerve-trunks due either to traumatic arterial spasm or to pressure beneath the deep fascia or to both, it must be borne in mind that often the clinical picture of nerve ischæmia is obscured by coincident mechanical injury to one or more nerves.

DISCUSSION OF INDIVIDUAL CASES

While space does not permit of a full discussion of all the cases, there are a few points which require special mention:—

Cases 1–7 can be regarded as more or less typical of ischæmic nerve lesions due mainly to pressure beneath the deep fascia, though in *Case* 6 there was evidence of traumatic arterial spasm.

Case 8 is of particular interest, for, although a lesion of the main artery of the limb was known to be present, signs of nerve damage were definitely related to the development of a large collection of blood beneath the deep fascia of the thigh. All the nerves lying within this fascial envelope were affected and the dissociation of sensation is well shown. The small areas of sensitivity to pin-prick in the proximal parts of the areas supplied by saphenous, musculocutaneous, and posterior tibial nerves are of special interest. The sensory loss in the area of the medial cutaneous nerve of the thigh is probably due to division at operation.

Case 9. The sequence of events here is difficult to explain, but in view of the history of pallor and of the definite presence of Volkmann's ischæmic contracture in the anterior tibial muscles, it is almost certain that traumatic arterial spasm was the main factor in the production of the nerve lesions.

Case 10. The presence of Volkmann's contracture in the biceps indicates that traumatic arterial spasm probably played an important part in the aetiology. The presence of a strong profunda brachii pulse can be correlated with the fact that the radial nerve was less affected than the other nerve-trunks.

Cases 11, 12. Though in both these the radial nerve had been severed, the lesions of the median and ulnar nerves were probably ischæmic in origin.

Case 13. The pathology here is interesting and not very clear. A possible explanation of

the contracture in the interossei would be pressure on the radial artery, possibly with spasm extending into the deep palmar arterial arch from which these muscles are supplied. A relatively intact circulation through the ulnar artery and the superficial palmar arch would maintain the nutrition of the more superficial muscles and the digits themselves. The nerve lesions were probably due to pressure ischæmia of the median and ulnar nerves at the wrist.

Case 14. The mode of onset of anæsthesia, the retention of voluntary power in all intrinsic muscles, and the type of sensory loss suggest that pressure ischæmia of the nerve-trunks rather than a block by direct pressure on the nerve-fibres was the underlying cause. The median was more severely affected than the ulnar.

Case 15. In one respect this was the most interesting of the series. The ischæmia was apparently of such a degree as to cause peripheral degeneration of all median and ulnar fibres supplying the hand, with the exception of the small-diameter 'slow' pain fibres. Even these were blocked for a few days, but conduction in them was soon re-established in superficial radial, median, and ulnar nerves in that order. Presumably recovery of other types of fibres in median and ulnar nerves will have to await regeneration. At present the unpleasant delayed response to pin-prick and firm pressure is most striking. Another point of interest is that at no time was the circulation in the hand much impaired, which supports the view that the confinement of the effects to the peripheral distribution is not due to a circulatory effect on the peripheral nerve-endings. The circumflex nerve has probably suffered direct damage.

TREATMENT

It will be obvious that once severe ischæmia of nerve-trunks has caused peripheral degeneration of the fibres, little can be done in the way of treatment apart from physiotherapy, splinting, etc. Recovery will be dependent upon the slow process of regeneration. In cases where the diagnosis is in doubt or where the signs of nerve-trunk ischæmia are thought to be due to the involvement of the nerve in scar tissue, exploration may be indicated.

It is reasonable to suppose, however, that in many of these cases peripheral degeneration of the nerves could have been prevented by *early* recognition of the condition and by appropriate surgical intervention designed to relieve the ischæmia.

The early management of cases of traumatic arterial spasm has been described by Griffiths, Cohen, Trueta, and others. It includes such measures as forcing the collateral circulation by raising the body temperature. Heating of the *unaffected* limbs, the affected limb being exposed to environmental temperature, has been found especially valuable (Learmonth, 1943). Oxygen therapy by the B.L.B. mask, the injection of antispasmodics such as papaverine, and aravertebral or intrathecal injections of novocain, have all been recommended, but in most cases early exploration of the artery with periarterial stripping and possibly arteriectomy is indicated.

In those cases where the limb is grossly swollen and tense, and where there is anæsthesia commencing in the digits and gradually spreading centripetally, it would seem to be well worth while to relieve tension in the limb by freely incising the deep fascia. Even when the peripheral pulse is present and though it may mean converting a simple into a compound fracture, this procedure would be justified considering how serious and prolonged is the disability resulting from ischæmic nerve lesions such as those described above. The use of the closed-plaster method in the presence of swelling and obvious circulatory disturbance would appear to be particularly dangerous.

SUMMARY

Fifteen cases are described in which multiple peripheral nerve lesions are considered to be due to traumatic ischæmia of the nerve-trunks.

The aetiology is discussed and the conclusion reached that the nerve ischæmia is due either to traumatic arterial spasm or to gross swelling and pressure beneath the deep fascia.

The relation of traumatic ischæmia of peripheral nerves to Volkmann's ischæmic contracture is discussed and some observations made on the latter condition.

The importance of prophylactic treatment in the early stages is stressed.

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RETAINED INTRAVENTRICULAR FOREIGN BODY

BY J. M. SMALL, MAJOR, R.A.M.C.

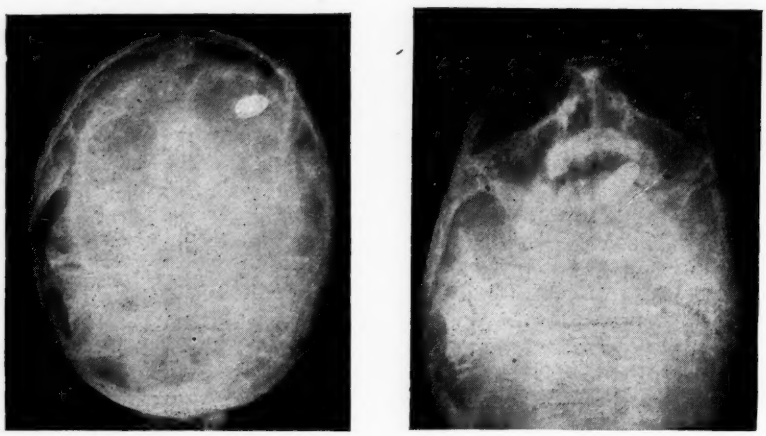
THE case presented is one of a retained revolver bullet within the left lateral ventricle following a left frontal penetrating injury. There are but few recorded cases of this type, and for this reason it has been thought justifiable to put on record this case history.

CASE REPORT

A 29-year-old officer received the following injury at about 5 p.m. on March 1, 1944: a 0·35 calibre pistol bullet entered through the left side of the hard palate, traversing the maxilla, maxillary antrum, the anterior portion of the left orbit, the anterior end of the supra-orbital plate, involving the frontal sinus, and then through the left frontal lobe to comminute the left frontal bone without involving the scalp. There was at no time any loss of consciousness, but on admission to a U.S. Naval Hospital at 10 p.m. on the same day he was reported to be rather drowsy, though otherwise to have no abnormal signs in the nervous system. There was no neck rigidity, his temperature was 99°, pulse-rate 62, respirations 22, blood-pressure 130/60. On the morning of March 2 his temperature had risen to 100·6° and lumbar puncture performed at the time showed a slightly blood-stained fluid at a pressure of 85 mm. In the evening his temperature was 100°. From 11 p.m. on March 1 until 9 a.m. on March 3 he received 11 g. of sulphadiazine.

On March 3, 1944, he was transferred to a military hospital for head injuries. At that time his only complaint was of headache and this of mild degree. He was fully conscious, rational, and perfectly orientated for time and place.

The left eyelids were œdematous and bruised; the dressing removed from this eye showed a small amount of pulped brain tissue on its surface. Fluid was escaping from between the eyelids, and this was possibly cerebrospinal fluid, but the swelling and tenderness of the eyelids was such that the eye could not be examined at this stage. There was a tender swelling in the left frontal region just anterior to the hairline, but the scalp was intact. In the left side of the hard palate was a clot covering a penetrating wound about the size of a sixpenny piece, with marginal pouting of the mucosa and powder staining of the palate in this vicinity. The patient was constantly sniffing, but there was no fluid escaping from the nostrils to suggest a cerebrospinal rhinorrhœa.



A B

FIG. 345.—Radiographs showing the mobility of the bullet within the frontal lobe before operation; the outward comminution of the frontal bone is well seen in B.

General examination was normal, the blood-pressure being now 130/80, temperature 98°, pulse-rate 60, respirations 18. There was no neck stiffness, and the Kernig test was bilaterally negative. The patient was right-handed. Cranial nerves were normal, except for left-sided anosmia. The vision and ocular movements of the left eye could not be ascertained. Power and co-ordination and all aspects of sensation were normal in the limbs. The deep reflexes were normal and equal, plantars bilaterally

downgoing, abdominals diminished on the right side. There was no dysphasia of any type. Mental state: Fully conscious and orientated. Mental arithmetic excellent; 100—7 test, correct in 50 seconds. Digit retention—6 forwards, 5 backwards. Cube test rapidly and correctly done.

Lumbar puncture showed mildly blood-stained fluid under a pressure of 140 mm.; protein 25 mg. per cent, without excess of globulin; R.B.C. 10,100, polymorphonuclears 35, lymphocytes 52 per c.mm. No bacteria were seen on smear and there was no growth on culture. Blood-count: R.B.C. 3,280,000, hæmoglobin 82 per cent, W.B.C. 23,600, of which 90 per cent were polymorphs.

Radiographs arriving with the patient showed the bullet lying in the left frontal region, low down on the supra-orbital plate, and a further series of radiographs on admission showed that this bullet had moved forwards and was lying apparently with its tip within the frontal sinus (*see Fig.* 346); while a submento-vertical picture showed that the bullet was mobile within the frontal lobe and had dropped backwards and medially when the head was in this position (*Fig.* 345). It also showed an outward comminution of the frontal bone, from which point the bullet must have ricocheted back into the frontal pole. The X rays also showed the track of the bullet, with fracture of the hard palate, dimmed left antrum, and fracture of the orbital floor and the orbital roof, involving the frontal sinus.

He was given 3000 units of A.T.S., and sulphadiazine, 2 g. 4-hourly by mouth, was continued.

OPERATION (11 p.m., March 3, 1944, 54 hours after receipt of the injury).—General anæsthesia—intratracheal gas, oxygen, and ether.

Before operation was commenced, while the patient was under the anæsthetic, the left eye was examined and was found to be completely disorganized, with loss of vitreous. It was quite obvious that evisceration would be necessary. Intravenous plasma and blood being used from the outset, a left frontal scalp flap was turned down, exposing comminuted bone and pericranium, with grossly contused cerebral tissue and clot pushing its way through the fenestrations. The pericranium around this comminuted area was encircled with the cutting diathermy and the lacerated pericranium and comminuted bone fragments were gently lifted away, exposing the traumatized brain protruding through a considerable dural laceration. The pulped brain was sucked away and then covered with a hot pack while bone was nibbled away around the margins of this irregular skull defect, until the dura was seen all the way round, except in the region of the supra-orbital margin, which was also grossly comminuted. Here the fragments of bone were allowed to stay in place because of their proximity to and association with the frontal sinus. A left frontal skull flap including the skull defect was then turned down as an osteoplastic flap hinged on the left temporal muscle, the bone flap going to the middle line. The torn margins of the dural defect were then trimmed and the dura further opened to give a view of the frontal tip right up to the sinus and falx. With this adequate exposure all contused brain tissue was then cleared out of a wedge-like cerebral laceration, the apex of the wedge pointing directly backwards more or less in the centre of the frontal tip. To obtain adequate room to examine the supra-orbital plate and to leave only healthy brain behind, the surgical line of incision was made about ½ cm. away from the margin of the brain laceration and this fringe of brain removed all round, right down on to the floor of the anterior fossa, so that only normal healthy brain remained. The floor and supra-orbital part were then inspected and were quite normal, the laceration in the dura only going down to the junction of the vault and frontal base beyond the frontal sinus.

No sign had been seen of the bullet. A good view was obtained right back along the base underneath the frontal lobe, and there was no evidence that it had escaped in that direction. The whole of the brain cavity was then palpated with a brain needle and at

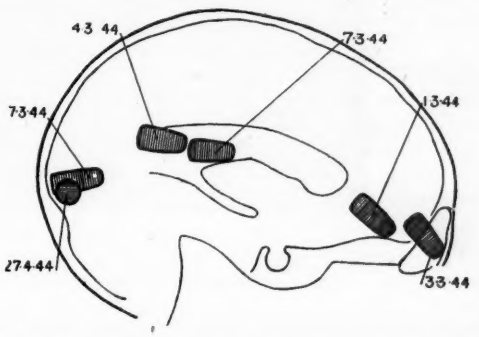


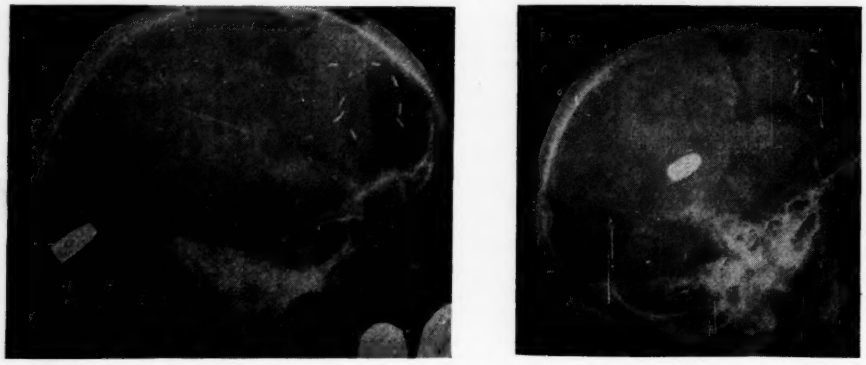
FIG. 346.—Diagram illustrating position of bullet at various dates.

one point leading directly backwards into the frontal lobe the brain needle simply fell in. This was obviously the track and was therefore followed with the sucker and brain retractors for 3–4 cm. The ventricle was never certainly seen, though at one time it seemed likely, because some fluid mixed with blood came up along the track and this may well have been cerebrospinal fluid from the ventricle. A troublesome bleeding-point was encountered at the bottom of this track, and it was felt that it would do more harm than good to search further for the bullet in that direction. It was therefore decided to have the patient X-rayed on the table to see what had happened to the missile; when this had been done it was obvious that the bullet had fallen back through the ventricle and was lying in the occipital horn (*Fig.* 346, March 4) and that at that time there was nothing to be done about it.

The possibility of turning the patient over and seeing whether the bullet would fall out was considered too precarious a thing to do after the considerable operation that he had already undergone. Four c.c. of 500 units per c.c. penicillin were injected down the brain track and presumably much of this entered the left lateral ventricle. The whole of the dural defect was then trimmed, and a piece of fascia lata sewn into place to close the dura completely. Next, the comminuted bone of the supra-orbital ridge was removed. This laid the frontal sinus wide open; its mucosa was removed and a piece of fascia lata sutured over the apex of the exposed frontal sinus to close it off further. The dura was sutured up all the way round the flap margin to the pericranium and the bone flap was sutured back into position, a gutta-percha drain being led out from the extradural space through the posterior burr hole of the flap and through the posterior limb of the incision. Sulphanilamide powder was added to all layers of the closure, including the brain cavity. The left eye was then eviscerated and the remaining cavity powdered with sulphanilamide and a light pack of vaseline gauze inserted.

At the completion of the operation a lumbar puncture was performed and 4 c.c. of 500 unit per c.c. penicillin were injected into the spinal theca.

The examination of this cerebrospinal fluid showed that it had a protein content of 175 mg., 24,600 R.B.C., 140 polymorphs, and 75 lymphocytes. No bacteria were seen on the film and none were grown on culture. Bacteriology of bone fragments removed at the time of the operation showed no bacteria from surface swab and no growth on culture.



A B

FIG. 347.—Four days after operation. A (brow-up) shows bullet in occipital horn. B (brow-down) shows that the bullet has moved forward to the region of the trigone of the ventricle.

Following operation the patient continued to have 2 g. of sulphadiazine 4-hourly until March 8, 1944, 66 g. in all being given. The day after operation he had considerable neck stiffness and a positive Kernig, was drowsy and incontinent, but there was no other discernible change in the physical signs in the nervous system. Lumbar puncture at that time showed a pressure of 160 mm., and the cerebrospinal fluid contained 420 mg. of protein, 14,500 R.B.C., 3100 polymorphs, and 55 lymphocytes. No bacteria on film and no growth on culture. Because of the persistence of neck stiffness and temperature to 100·6°, a further 4000 units of penicillin were given intrathecally by lumbar puncture on March 6, at which time the cerebrospinal fluid protein had fallen to 45 mg. per cent, but its cellular content was still high (7800 R.B.C., 4500 polymorphs, and 62 lymphocytes per c.mm.). Again no bacteria were found on film or culture. From that time on, he made an uninterrupted recovery. He became rather euphoric, but apart from this there seemed to be no evidence of intellectual impairment. The operation wound healed by first intention (*Fig.* 349). The bullet wound of the palate healed rapidly without any surgical treatment whatsoever, and in the end there was an almost unidentifiable scar in the region of the penetration. No treatment was ever required for the antrum and the orbit healed and granulated satisfactorily.

On March 5, 1944, X-ray screening showed that the bullet remained in the occipital horn of the ventricle. On March 7 screening showed that the bullet was mobile within the ventricle, moving forward from the occipital pole to about the trigone of the ventricle when the head was put in the brow-down position (*Fig.* 347). It could not be brought farther forward than this, nor would it go into the temporal horn. By March 18 the bullet was in the occipital horn, lying rather transversely and fixed in this position, no movement occurring, whatever the position of the head. Strangely enough, this bullet appeared

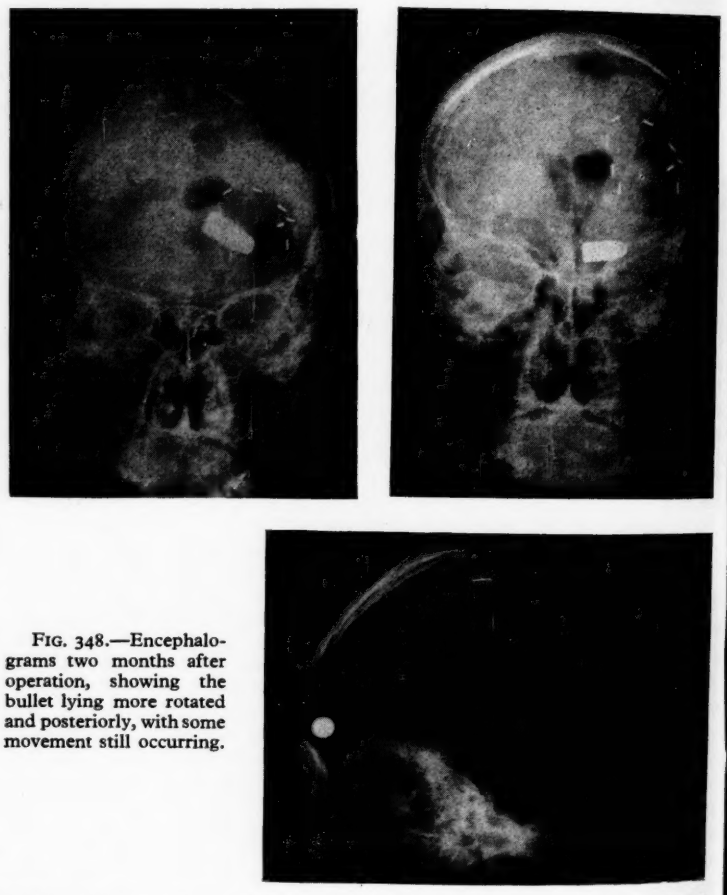


FIG. 348.—Encephalograms two months after operation, showing the bullet lying more rotated and posteriorly, with some movement still occurring.

to be very far back in the occipital pole, either in a large occipital horn or actually pushing its way out from the ventricle into the occipital lobe proper.

On March 20 screen testing of the right eye showed that there was 6/6 vision and a full field.

By April 26 the X-ray appearances of the bullet were that it was lying even more transversely and farther posteriorly, apparently almost on the occipital bone itself. A cisternal encephalogram performed on April 27 showed that the bullet was lying in the occipital lobe, and although there was some dilatation

FIG. 349.—The patient at the time of encephalography

of the left lateral ventricle in its entirety, with some distortion of the anterior horn, the bullet had apparently left the ventricle itself and there was no visible dilatation of the occipital horn to suggest that it was lying in a prolongation from the ventricle. Slight movements of the bullet within the occipital lobe still appeared to be taking place as judged from the films (*Fig.* 348). The cerebrospinal fluid taken at this time from the cistern had a protein content of 20 mg. per cent and no cells. Visual acuity remained at 6/6 in the right eye and the field was full and unchanged. On full intellectual testing on May 3, 1944, the patient showed some slight evidence of intellectual impairment and a mild, but definite, euphoric state.

DISCUSSION

There is a similar case recorded by Campbell, Howard, and Weary (1942), in which a lead buckshot entered the left lateral ventricle in the frontal region and a week after the injury the bullet was shown lying deeply in the left frontal lobe in the middle line. On X-ray examination two weeks later it was seen to be in the occipital lobe. Movements of the head allowed this bullet to roll freely into the frontal lobe and even into the temporal lobe, during which movement no disturbance of any description was noted or complained of by the patient. Two weeks after the initial injury there was a sudden onset of headache, vomiting, and neck stiffness, and there were 210 cells in the cerebrospinal fluid, 65 per cent polymorphs. At this time X rays showed that the bullet had become fixed in the occipital lobe. This bullet was removed from the occipital horn by an occipital flap without consequent disturbance of speech or visual fields. In spite of pleocytosis the cerebrospinal fluid had always been sterile, and the bullet when it was removed proved to be sterile on culture.

These writers mention another case, of Dandy's, in which it is said that there were such severe symptoms that removal was necessary; but they also mention one other case in which a small roundshot wandered freely from the lateral ventricle to the lower part of the spinal canal without giving rise to any symptoms or discomfort. Geoffrey Jefferson (1918) records a case of movement occurring in a bullet in the cerebellar hemisphere. This bullet moved backwards by 2 cm. towards the occipital bone, and turned round as well, during the course of a few days when it was studied radiologically. On removal it was found to be within a sterile abscess cavity. He also draws attention to the fact that bullets experimentally introduced into the cerebral hemispheres in animals always tend to sink towards the base, and suggests that these movements are due to the high specific gravity of the bullet as compared with brain tissue. He further suggests that movement will be encouraged by surrounding softened brain tissue or abscess formation and pulsation of the brain itself, the rotation which is observed being possibly due to the different weight in different portions of the missile and to the different resistances that it may meet—for example, a portion of it being resisted by a leash of blood-vessels.

The involvement of the ventricle in penetrating wounds of the brain is always of serious significance, and in a review of cases from the last war Cushing (1918) reported 30 cases of ventricular penetration of which only 8 recovered. These 8 were from a series of 14 in which the ventricle had been penetrated by bone fragments and not by the missile itself. All the other 16 cases, in which the projectile either entered or passed through the ventricle, were fatal. Involvement of the ventricle in penetrating wounds has always seemed to carry a mortality of 60 per cent or above, and in some of the recorded series it has been as high as 100 per cent. In this connexion it should be remembered that in the present case prophylactic chemotherapy with penicillin and sulphadiazine was employed.

This case is particularly interesting because the missile was of low velocity and was unlikely to be sterile; even so, after a period of 54 hours its entrance into the ventricle only caused a mild disturbance of cerebrospinal fluid chemistry and cytology. Most other cases recorded have eventually led to removal of the missile because of meningeal infection or irritation. There seems to be no indication for removing the bullet in this case—in fact, there is a definite contra-indication, in that the patient has only one remaining eye, and operative interference would

almost certainly result in a residual field defect and possible speech disturbance.

SUMMARY

A case is described of ventricular penetration by a revolver bullet which was at first freely mobile within the ventricle and then became lodged within the occipital horn. Spontaneous backward movement and rotation then occurred within the occipital lobe.

I wish to thank Lieut.-Colonel C. W. Greenway, R.A.M.C., for permission to publish this paper, and Brigadier Hugh Cairns for his help in its preparation.

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ASEPTIC ANASTOMOSIS IN GASTRECTOMY

BY CHARLES A. PANNETT

FOR many years aseptic anastomosis of the colon has established its place as a sound method of union for this part of the bowel. Its principles have not often been applied to the joining of the jejunum to the stomach after gastrectomy. The contents of these regions of the alimentary canal having very few organisms in them, there has not seemed to be the same urgency for the prevention of soiling with the gastric or jejunal juice. Moreover, the great vascularity of the stomach has aroused fears about the efficacy of hæmostasis in the closed method. However, W. Wayne Babcock (1942) has described the use of an aseptic method of anastomosis. For the last three-and-a-half years I have performed gastrojejunal anastomoses exclusively by a one-clamp technique. The advantages have been very great. There is no soiling, the method saves time, no encumbering hæmostatic compression clamps are necessary on the stomach or jejunum, and there is no loss of blood during the actual stitching of the viscera. Nor in all this time has there been a single case of hæmorrhage after operation. The post-operative course appears to have been smoother.

FIG. 350.—General plan of operation.

The operation is carried out in the following way: The greater and lesser omenta having been separated from the two curvatures of the stomach and the ulcer freed from its adhesions, the duodenum is cut across beyond the ulcer if it is in the duodenum itself, or just beyond the pylorus in the case of a gastric ulcer or carcinoma, and the distal end sewn up. The freeing and suturing is carried out according to the methods I have already described. The stomach is then thrown over to the left of the patient and the line of section selected. The first part of the

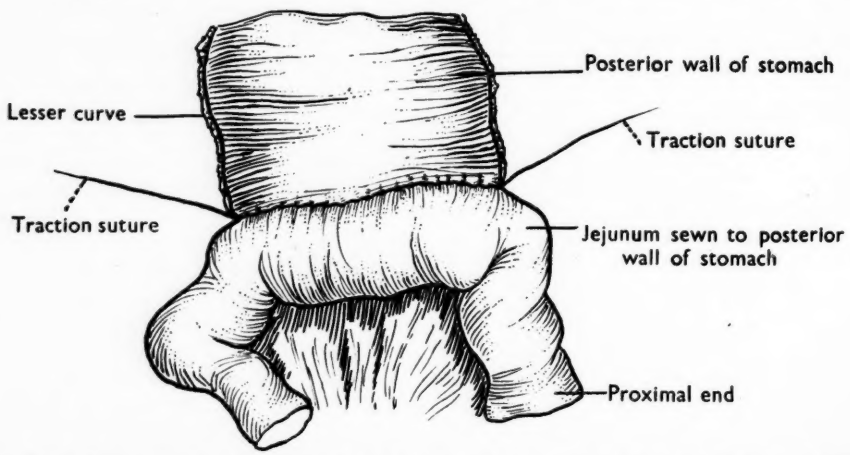

FIG. 351.—First stage of anastomosis. The stomach has been turned over to the left of the patient. Jejunum sutured to posterior wall of stomach by two rows of sutures. View as seen by surgeon standing on right side of patient.

jejunum is then brought up in front of the colon and at a distance of 5 or 6 in. from the duodeno-jejunal flexure sutured to the stomach just

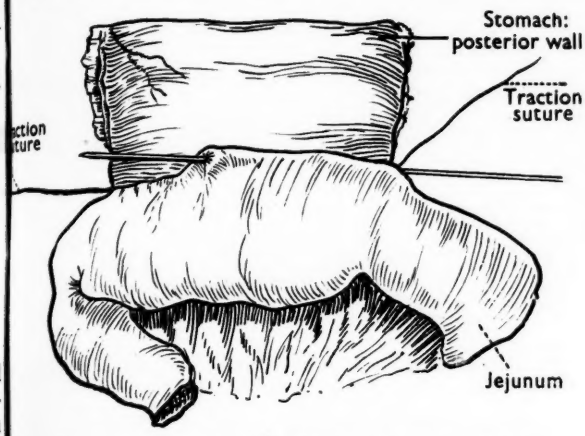

FIG. 352.—The wall of the jejunum has been pulled up in a fold by transfixing it with a wire.

proximal to the selected line with two rows of a continuous thin catgut stitch. The proximal end of the jejunum is at the greater curvature. I have tried interrupted cotton stitches, but all the advantages lie with thin catgut as suture material, used as a continuous stitch (*Fig.* 351). A pointed wire (a pointed steel knitting needle or a Kirschner wire) is then thrust into the jejunum and out again as depicted in *Fig.* 352, in order to drag up a fold of jejunal wall. This step is important. Unless this is done it is very easy to pull the muscular coat up away from its loosely attached mucosa and fail to penetrate the lumen of the bowel when the fold in the clamp is burnt off. Two of my duodenal clamps

a little distal to where it has been sewn to the jejunum (*Fig.* 353). A soiling-prevention gastric clamp is then applied to the stomach distal to the two special duodenal clamps, and the stomach and jejunal fold beyond these latter clamps removed with a cutting diathermy current. A coagulating current is then used to ensure hæmostasis along the cut surface of the tissues in the grasp of the clamps. That clamp on the lesser curvature of the stomach is then loosely oversewn with a continuous suture beginning at the lesser curvature, the clamp removed, and the stitch tightened and carried back as a second row to its beginning. This second row usually passes

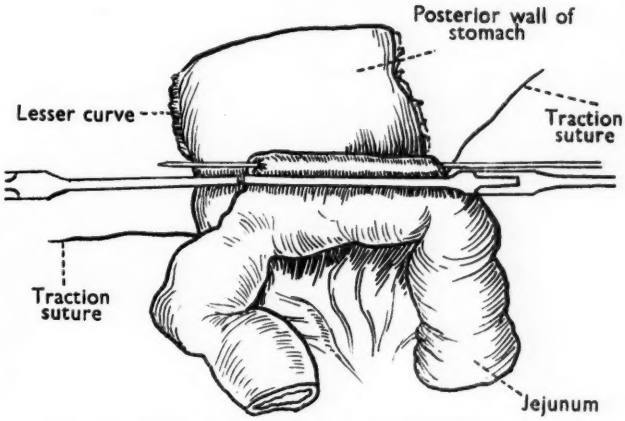

FIG. 353.—The stomach at left, and stomach with jejunal fold at right, have been seized in clamps.

between anterior wall of stomach and jejunum. In this way the upper part of the cut end of the stomach is closed. For reasons I have mentioned elsewhere it is important that the jejunum should

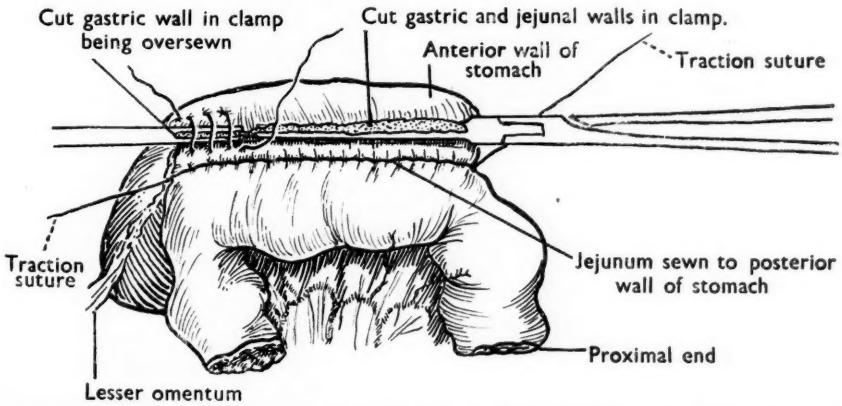
Cut gastric wall in clamp being oversewn
Cut gastric and jejunal walls in clamp.
Anterior wall of stomach
Traction suture
Traction suture
Jejunum sewn to posterior wall of stomach
Proximal end
Lesser omentum

FIG. 354.—The stomach and jejunal fold distal to the clamps have been removed by diathermy cautery. The part of stomach in clamp at lesser curve is being sewn up.

are then taken. Any narrow clamp made so that slipping of the bowel from its grasp is prevented and with blades 2½ in. long will do. One is made to seize the two walls of the stomach and the fold of jejunum; the other is applied to the remaining part of the stomach between the tip of the first clamp and the lesser curvature

be attached to this part of the stomach (*Figs.* 354, 355). The anterior wall of the stomach is now sewn to the anterior wall of the jejunum over the clamp which has the four layers of visceral wall in its grasp (*Fig.* 356). The clamp being removed, the stitch can be pulled tight and the anastomosis effected. In doing this,

to avoid cockling the suture line the two ends of the loose stitch must be held absolutely horizontal. I usually knot the beginning of the segment of jejunal wall is removed in place of the linear incision in the ordinary anastomosis. Whether it is due to this or to the fact that there

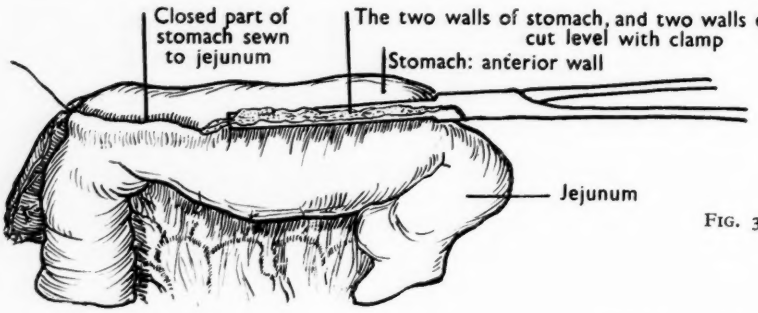
Closed part of stomach sewn to jejunum
The two walls of stomach, and two walls of fold of jejunum cut level with clamp
Stomach: anterior wall
Jejunum

FIG. 355.—Upper part of cut end of stomach closed and sewn to jejunum.

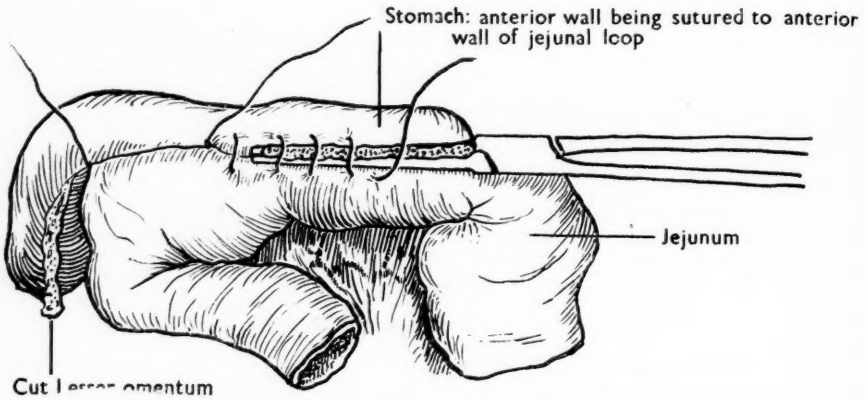

FIG. 356.—Anterior wall of stomach being sewn to anterior wall of jejunum over the clamp.

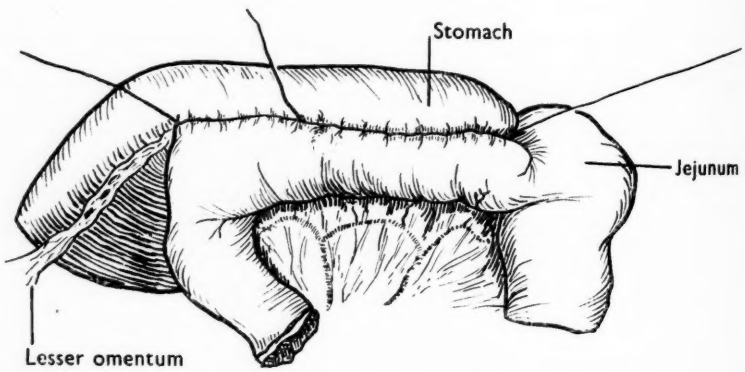

FIG. 357.—Anastomosis completed by additional row of stitches.

stitch at the left as in the figure, and find no difficulty in pulling the suture taut provided the individual mattress stitches are made parallel to the line of junction. The suture is carried back to its beginning to make a second row and complete the junction (*Fig.* 357). The walls of the viscera immediately separate and form the communication. It may be that the passage of gastric contents is made easy because an oval is less soiling in the neighbourhood of the anastomosis, convalescence is smoother with this method of union than that usually performed.

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RIGHT RETROPERITONEAL DIAPHRAGMATIC HERNIA

By N. R. BARRETT

SURGEON TO OUT-PATIENTS AT ST. THOMAS'S HOSPITAL, LONDON

THE details of this case are presented because the lesion from which the patient suffered is rare and because it raises some interesting points in diagnosis and treatment.

CASE REPORT

HISTORY.—A shopkeeper, aged 52, was admitted to St. Thomas's Hospital in March, 1944.

In December of the previous year he had had an attack of diarrhœa, pains in the lower part of the right chest, and headache. His doctor found no physical signs to account for these symptoms, but there was dullness to percussion and diminished air entry at the back of the right chest. Lobar pneumonia was diagnosed. The patient was kept in bed for three weeks and given a course of sulphonamides, and during this time he was ill and run-down, but had neither cough, sputum, nor pyrexia. He stated that when the time came for him to get up he felt very weak and that he suffered from dyspnœa upon the slightest exertion. During the early months of the year his condition improved considerably, but he was eventually admitted to the hospital complaining of abdominal pain and dyspnœa on exertion.

ON EXAMINATION.—The patient was a rather stout man of Russian extraction. His general condition was good; he was apyrexial and the pulse and respiratory rates were normal. The only previous illnesses, which he admitted at the time, were an attack of typhus fever 24 years previously, and indigestion for which he had been treated on and off for a long time. The latter had never been very serious, but a diagnosis of duodenal ulcer had been made on several occasions, and radiographs had been taken at other hospitals. The details of these investigations were not available, but neither regulation of his diet nor the use of alkalis had ever had any effect upon the attacks of abdominal pain, and he regarded the 'indigestion' from which he was suffering at the time of his admission as "just another bout of the old trouble". Physical examination was entirely negative except for signs in the lower part of the right chest which were indicative of a solid mass lying posteriorly at the base of the thorax. The signs also suggested that a thin shelf of lung tissue was interposed between the mass and the chest wall, and a provisional diagnosis of carcinoma of the lung was made.

No sputum was available for examination and the Wassermann reaction was negative. Radiography of the thorax revealed an opacity at the base of the right chest which merged with the shadow of the diaphragm and the liver below, and which had a definite upper edge running transversely across the lower third of the right lung field (*Figs.* 358, 359). The films indicated that the lesion was probably extrapulmonary in position and a diagnostic pneumothorax was induced to settle this point. The following additional information was provided by subsequent radiographs: the mass was extrapulmonary; it lay at the back of the pleural cavity in close proximity to, but separated from, the chest wall; it was lobulated and showed areas of varying density; the lower edge merged with the diaphragm and was quite separate from the lung; there was no evidence of calcification (*Fig.* 360). A follow-through barium meal demonstrated "some deformity of the duodenal cap (which was somewhat higher in the abdomen than usual) considered to be due to adhesions outside the bowel. No evidence of duodenal ulceration was seen".

DIAGNOSIS.—The information at our disposal suggested that the differential diagnosis lay between a diaphragmatic hernia, a tumour of the diaphragm, or, possibly, a tumour of the chest wall. The first of these alternatives was rejected because no part of the intestinal canal could be demonstrated by X rays to be abnormally placed. This was not a valid reason for discarding the diagnosis of hernia, because it overlooked the fact that solid viscera can pass into the chest without being accompanied by intestine, and it is a pity that pyelograms were not made.

The second possibility was a tumour of the diaphragm. This term is used in its widest sense to include cysts and granulomata. The condition is rare, and Robson and Collis (1944) state that only 21 examples have been recorded in the literature—in a more recent paper Barrett and Thomas (1944) describe an additional case, in which operation was successfully performed by Mr. C. Price Thomas. The points against such a diagnosis were the size of the lesion, and its position right at the back of the muscle.

Chondroma and chondrosarcoma of the chest wall were ruled out because, both clinically and by X rays, the mass did not lie in continuity with the ribs.

Being at a loss for a definite diagnosis, thoracoscopy might well have been performed, and if this operation had been done it is certain that the lesion would have been considered to be a large lipoma.

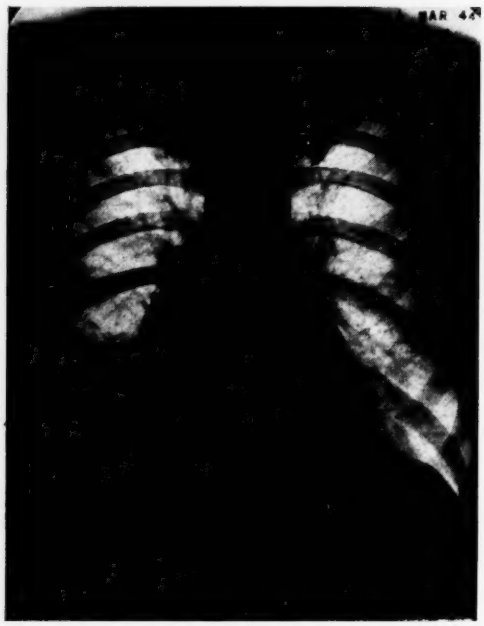
The patient was advised to have a thoracotomy, but he was most anxious to avoid operation, and at this point he volunteered the information that he had been wounded in the right chest 26 years previously. This was the first time he had spoken of this injury, and he now admitted that he had been stabbed in the back by a narrow-bladed knife at the time of the Russian Revolution. The injury had not been serious and he had only been confined to bed for a short time; no special treatment had been given and convalescence was uneventful. Upon careful examination of the skin of the back a tiny scar, lying over the 10th rib at a distance of 3 in. from the midline, could still be detected.

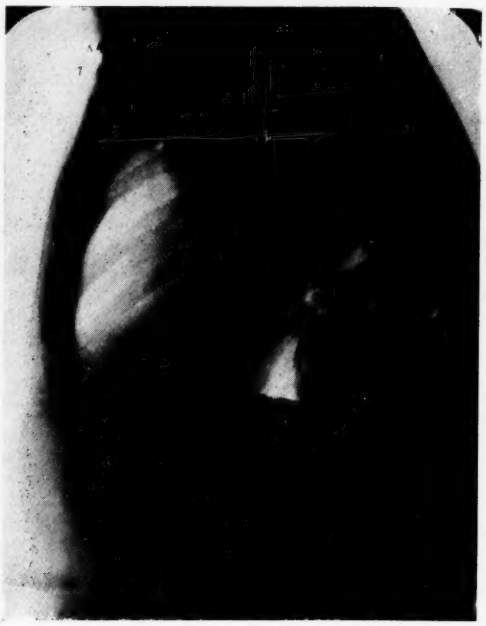
OPERATION.—Thoracotomy was performed at Horton War Hospital. Dr. M. D. Nosworthy administered a general anæsthetic. Controlled respiration was used, and a blood transfusion was set up as a prophylactic measure.

The chest was opened by the usual intercostal incision and the pleural cavity was free from adhesions. A large mass of lobulated fat was immediately discerned upon the upper aspect of the diaphragm in the position indicated by the X rays. A little dissection revealed that the 'tumour' was a retroperitoneal diaphragmatic hernia containing the right kidney surrounded by the perinephric fat. There was no hernial sac. The aperture in the diaphragm

was about 3½ in. long and, after the hernia had been reduced, this was repaired in the usual way with two rows of thread sutures. At the conclusion of the operation the chest was closed in layers and the lung was re-expanded by aspirating the air in the pleural cavity. Convalescence was uneventful and, at the time of his discharge from the hospital, the patient had no symptoms.

It is interesting to note that although the upper half of the kidney lay in the right pleural cavity neither the suprarenal body nor the duodenum were seen at the operation.





FIGS. 358, 359.—Anteroposterior and lateral radiographs of the chest, taken at the time of the patient's admission to St. Thomas's Hospital.

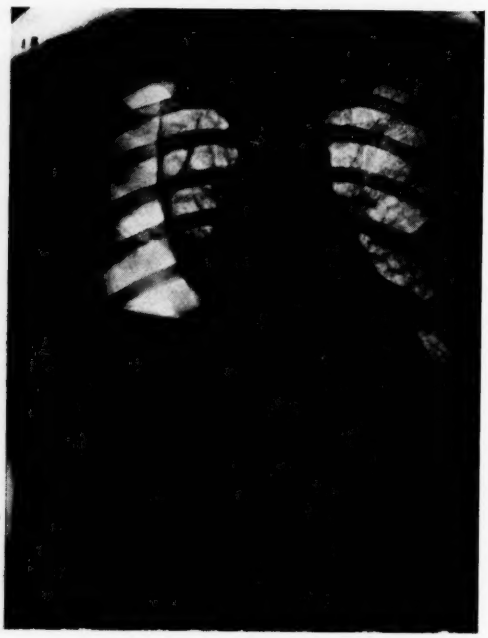


FIG. 360.—Radiograph taken after induction of a diagnostic artificial pneumothorax, showing the lung to be quite free from the mass.

The 'indigestion' from which the patient had suffered for so many years was presumably due to distortion of the duodenum occasioned by the abnormal position of the kidney. Six months after operation he stated that both the indigestion, and the dyspnœa for which he was admitted to the hospital, had been cured.

COMMENT

Diaphragmatic hernia is a subject which has been dealt with by many writers (Barrett and Wheaton, 1934; Bowen, 1938; Donovan, 1938; Dunhill, 1934; Harrington, 1942; Hedblom, 1925; Morton, 1939) in recent times, but the above case raises several points which are not generally discussed in these papers:—

1. Why is the lesion more common upon the left side of the body than the right? Bradley (1930) states that the proportion of left-sided to right-sided cases is at least 12 to 1, and the size and position of the liver, in the adult, is not sufficient to account for this by itself. The explanation varies according to the type of hernia under consideration.

In cases due to congenital malformations of the diaphragm Hume (1931) states that there is a strong probability that the defect occurs at that time in the growth of the fœtus when the mobile parts of the alimentary canal are undergoing spontaneous reduction, from the hernia in the umbilical cord, into the abdominal cavity—that is, at about the eighth week of intra-uterine life, when the embryo is approximately 20–22 mm.

long. (This supposition is supported to some extent by the observation that the majority of the cases have a hernia at birth, and about 75 per cent die before the age of one.) If the development of the diaphragm is normal the pleuroperitoneal membranes should have formed and closed the pleuroperitoneal canals, on both sides, by the time physiological reduction of the umbilical hernia occurs. The two canals do not, however, close at the same time—that on the right should seal off at the 17-mm. stage and that on the left at the 20-mm.—and, in consequence, there is more than a chance that the process may not be complete on the left side at the critical moment. There are other factors which favour a left-sided lesion; the liver is entirely a right-sided viscus at this stage of development; the stomach has reached its adult position below the diaphragm; and the left lung is considerably smaller than the right, and so it is not only easier for a left-sided hernia to occur, but there is more space in which it can be accommodated. These explanations are also relevant to another group of congenital cases—namely, those which occur through the dome of the diaphragm posterior to the left tendinous trefoil.

Hernia through the œsophageal hiatus is regarded by most authorities as being due in the first instance to a congenital malformation of the aperture (Hume, 1931; Harrington, 1938). This malformation may take the form either of an unduly large gap or of an absence of one of the crura of the diaphragm. Harrington (1938), who believes that such herniæ are congenital, admits that in his large series of cases the average age at which symptoms first occurred was between 50 and 60 and it may be that the real aetiological factor is that mentioned by Allison and Johnstone (1943). These authors point out that in infancy and in old age there is, normally, a discrepancy between the diameter of the œsophagus and the hiatus in the diaphragm, and through this space the stomach is apt to be drawn up into the chest. Hume (1931) expresses the opinion that such herniæ protrude commonly from the mediastinum into the right pleural cavity, and is satisfied that the sac into which the abdominal viscera pass is in reality a remnant of the lesser peritoneal sac, which, in the embryo, extends upwards as far as the hilum of the right lung. My own experience is that left-sided paraœsophageal hernia is the commoner variety, and the simplest explanation of this is that the hiatus lies to the left of the midline, and the stomach and the spleen, which are the usual contents of the hernia, are also disposed on the left side of the abdomen.

The chance of the diaphragm being perforated by traumatic accidents is equal upon the two sides of the body, but the risk of a hernia developing subsequently is greater upon the left than the right because the liver seals the gap with greater certainty than does the stomach, the spleen, or the colon. It is a mistake, however, to imagine that the liver cannot herniate through a small perforation of the diaphragm. In life it is a pliable viscus and either a small part of it or, in the case of extensive injury, a whole lobe can pass into the right pleural cavity. In the case of diaphragmatic tears due to crush injuries of the lower chest or the abdomen, the risk of hernia is also greater on the left because the liver acts as a buffer which absorbs the bulk of the pressure changes at the moment of crushing and, in consequence, the right diaphragm is not so apt to be ruptured as the left.

2. The case described above occurred as a result of a stiletto wound sustained at the time of the Bolshevik Revolution in Russia. The scar in the skin was very small and it is probable that the original tear in the diaphragm was of similar dimensions, but, at operation, the hole in the muscle measured about $3\frac{1}{2} \times 2\frac{1}{2}$ in. This simply means that diaphragmatic hernia behaves in a manner comparable to other types of hernia, in that once the process had been initiated there is a tendency for the lesion to become larger and larger. The importance of the matter lies in the fact that many soldiers sustain small penetrating wounds of the diaphragm, and, as neither the signs nor the symptoms of a hernia may be present during the period of convalescence, the lesion often reaches considerable proportions before its presence is suspected. During this war I have done thoracotomies on patients soon after a chest wound has been sustained and on several occasions have observed a tiny unsuspected perforation of the diaphragm. Within a few hours such a perforation is generally sealed off by a small plug of omentum, which may cure the condition, but which is also likely to act as the apex of a hernia (*Figs.* 361, 362). In other cases serial radiographs have shown that, as time passes, more and more of the abdominal contents are sucked into the pleural cavity, partly on account of the relatively negative pressure therein, and partly because of the piston action of the muscle itself. This sequence of events accounts for the cases in which a large diaphragmatic hernia is discovered for the first time many years after a patient has been wounded, and it follows that any patient whose diaphragm has been traversed by a small missile should be watched as carefully as those whose hernia is diagnosed early. I have seen two patients who sustained 'minor' thoracic wounds in the last war, who had no symptoms at first, but who after a lapse of some years developed abdominal pains which were diagnosed as 'hysterical'. Both were suffering from large diaphragmatic herniæ.

3. The diagnosis of diaphragmatic hernia is generally based upon the X-ray demonstration of some part of the stomach or the intestines in the thorax. The fact that solid viscera, such as the kidney, the liver, or the spleen may be the only ones to pass through, is apt to be overlooked. The normality of a follow-through barium meal does not in fact negative the diagnosis, although it renders it unlikely. Nor is it true to suppose

that the mass of the liver will prevent intestines entering the right pleural cavity. It has been stressed by many authorities that the intestines may occupy any type of right-sided diaphragmatic hernia and I do not doubt that, if the case at present reported had been allowed to persist, the duodenum would have passed upward into the chest as an hernia 'en glissade'. Duodenal symptoms were already present.

In this connexion one other point may be mentioned. Routine X-ray examinations of the abdomen reveal the fact that the hepatic flexure of the colon may be insinuated in a normal individual between the dome of the right diaphragm and the upper margin of the liver (*Figs.* 363, 364). In such a case it would be particularly easy for the colon to enter the right pleural cavity if the diaphragm were to be perforated, and I have

FIG. 361.—Radiograph of the lower chest and abdomen of a soldier, wounded on the previous day. A small fragment of metal, which can be seen lying between the first and second lumbar vertebræ, entered the left chest in the axillary region, passed through the lung and the diaphragm, without causing much clinical disturbance, and lodged in the abdomen. Note that the diaphragm appears to be intact at the time this film was taken.

FIG. 362.—Radiograph of the same patient taken two months later and showing that a small omental hernia has occurred. The mass projects through the wound, in the dome of the left diaphragm, into the left pleural cavity. This hernia, which was causing pain, was cured by operation.

FIGS. 363, 364.—Anteroposterior and lateral films of the chest and upper abdomen of a woman showing the colon to be lying between the liver and the dome of the right diaphragm. This state of affairs was not causing any trouble, and the patient had been admitted to hospital for a totally different complaint.

information of a patient in whom the hepatic flexure herniated through a right open pneumothorax which occurred as a result of a wound.

4. The most unusual feature of this case is the fact that the right kidney and the perinephric fat migrated upwards into the chest, and although Harrington (1938) says that this may occur in some cases with a congenital deficiency of the posterolateral part of the diaphragm, I have not found it described as a result of trauma on the right side.

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EXPERIMENTAL SURGERY

THE CONTROL OF HOSPITAL INFECTION OF WOUNDS

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THIS survey was undertaken in the light of recent accounts of hospital infection of wounds (Miles et al., 1940; Spooner, 1941) to extend the observations by McKissock, Wright, and Miles (1941) of the effects of a revised dressing technique on the incidence of added hospital infection. As in previous investigations, our survey was made in terms of *Str. pyogenes*, but our observations differ from them in that we have examined wounds and personnel for the presence of *Staph. aureus* as well, and have tried to assess the magnitude of the reservoirs of both these infecting organisms in the environment.

METHODS

Bacteriological.—Wound swabs were cultivated aerobically and anaerobically on blood-agar, and in cooked-meat medium, which was plated on blood-agar after 2 days and incubated anaerobically. We have found this addition to direct plating to increase the number of swabs from which pathogens are isolated by 7 per cent. Nasal swabs were cultivated aerobically on blood-agar and throat swabs anaerobically on gentian violet blood-agar (Garrod, 1942). We have confined the term *Staph. aureus* to coagulase-positive staphylococci, and *Str. pyogenes* to beta-hæmolytic streptococci belonging to Lancefield's group A. All streptococcal strains isolated were typed by Dr. Elizabeth Topley of the Emergency Public Health Laboratory, Oxford, using Griffiths's (1935) method. By this means we had hoped to demonstrate the spread of infection among throats and wounds, and the addition of new types to wounds already infected with streptococci. But, owing to the diversity and serological irregularity of the strains isolated, it was not possible to make full use of the results obtained.

General.—We studied a 28-bedded male ward, used mainly for patients with septic wounds, for a few patients with uninfected wounds, and some with concussion.

Most of the wounds were dressed daily by the Sister and staff-nurses; the dressings were usually carried out in a side room off a corridor leading from the ward. Our study extended from Aug. 12, 1942, to June 2, 1943, divided into three periods: (1) Aug. 12–Oct. 20 (70 days), a control during which no alterations were made in the established ward routine; (2) Oct. 21–Nov. 30 (41 days), during which certain structural alterations were made in the dressing room, and the nursing staff were trained in a new routine, based on that suggested in the *M.R.C. War Memorandum No.* 6. The training was completed by the end of November. (3) Dec. 1–June 2 (184 days)—a test period to observe any changes in the incidence of added infection following the reorganization. During part of this period the test was complicated by the fact that some patients were treated with penicillin.

The comparison of results from a preliminary control period with those from a subsequent

period, as a means of judging the effect of a changed routine, will be valid only if, among other things, the risk, or 'load', of infection in the environment and the concentration of susceptible wounds among the patients is similar.

Accordingly, we have estimated the average daily magnitude of the reservoirs of infection in terms of carriers of *Staph. aureus* and *Str. pyogenes* both in the upper respiratory tract and in wounds. If it is assumed that the susceptibility of the wound to an added *Str. pyogenes* or *Staph. aureus* is constant throughout its history, a simple estimate of the population of wounds at risk during any period may be made by counting the number of occasions upon which added infection could have been observed by our particular method of examination (*see below*). The variations of susceptibility with wound age are not known, though it is possible that a long-established wound may be more resistant than a recent one. We have not, however, been able to follow enough wounds for the long periods necessary to test the possibility. Most of the patients who acquired an added infection did so during the first three weeks after admission. Since the average stay in the ward of, for example, the patients without streptococcal infection was 4·4 weeks, and only 7 of 71 patients stayed longer than 7 weeks, our results will not be unduly weighted by the contribution to the data of long-established wounds whose resistance may differ grossly from that of more recent wounds; little difference in our results would be made by ignoring patients followed more than 7 weeks.

As a routine we swabbed the nose and throat of all patients, and the wounds of such as had wounds, on the day of admission and subsequently once a week. The noses and throats of the nursing staff were swabbed weekly. We made a limited number of observations on other reservoirs of infection (e.g., air, dust, etc.) during the autumn of 1942.

DEFINITIONS AND CRITERIA ADOPTED

Added Infection.—The term 'added infection' is used in this report to denote a new-appearing infection (i.e., one which appears in the wound subsequent to the patient's admission to the ward), which we believe to result from the introduction of bacteria from the hospital environment. Since we do not know how long an organism latent in the wound at the time of admission might take to develop sufficiently to be recognized on culture, we have required that, before a wound can be considered free from either of the two indicator organisms, two consecutive routine cultures, with at least 5 clear days between them, should be negative. These two negative swabs we term 'qualifying swabs', since any wound so characterized qualified for admission to the group of wounds at risk of infection with *Staph. aureus* or *Str. pyogenes*. It is obvious that every wound must on admission undergo a qualifying period, and an infecting organism newly appearing in this period, though in fact it may have been added, cannot by definition be considered as such. If a wound loses an infection and yields the two negative qualifying swabs, it is again classed as a wound at risk.

Reference to *Fig.* 365 will make the definition of the qualifying swab clear. In Example 1 the negative admission swab A is also a qualifying swab, since 5 days elapse before the second swab, which is thus the second qualifying swab. The wound at the third swabbing R1—taken 14 days after admission—qualifies as a wound at risk. In Example 2 the admission swab cannot be used as a qualifying swab, since there are only 2 clear days before the weekly routine swab; here the wound is 18 days old at the third swabbing. Thus two qualifying swabs imply at least 7 days' freedom from infection. (Q1 — Q2 in *Ex.* 1; A — Q2 in *Ex.* 2).

The added infection rate is expressed as infections per number of wound-weeks at risk, and is calculated as follows. If R1 (*see Fig.* 365, *Ex.* 1) is positive, the rate is 1/1; if R1 is negative but R2 positive, it is 1/2, etc. So that for any wound showing added infection, the rate is $1/(a + 1)$, where a is the number of negative swabs observed after the qualifying period. The added infection rate for all wounds at risk is $n/(\Sigma a + n)$, where n is the number of added infections observed, and Σa the total number of negative from all qualifying wounds.

It should be noted that, as far as streptococcal infections are concerned, qualification is based solely on the absence of *Str. pyogenes*; the presence or absence of *Staph. aureus* is for this purpose immaterial. Similarly, the *Staph. aureus* infection rates are calculated independently of *Str. pyogenes* infection.

Load of Infection.—We have assessed the load of infection in the ward in terms of the average daily number of patients carrying *Staph. aureus* or *Str. pyogenes* in the upper respiratory tract or wound.

Swabs being taken, as a rule, at weekly intervals, we have had to make certain assumptions about the persistence of infection and of the carrier state (*see Fig.* 365). Where a positive swab was followed by a positive with no more than 10 days' interval, the whole of the intervening period was considered positive. Likewise, with two negative swabs, the whole period was considered negative (*Ex.* 3 and 4). Where a positive was followed by a negative with no more than 10 days' interval, half this interval was considered positive—half a day being counted as one (*Ex.* 5). If the interval was longer than 10 days, 5 days after a positive swab was the longest period assumed positive (*Ex.* 6). In no case was a patient assumed to be positive before the day of a positive swab taken during his stay in the ward (*Ex.* 7). When a positive swab had been received from a wound, persistence of the same

organism on a plaster covering the wound was reckoned as a positive source, since the presence of microbes on the surface of the plaster clearly constitutes a risk to other patients in the ward.

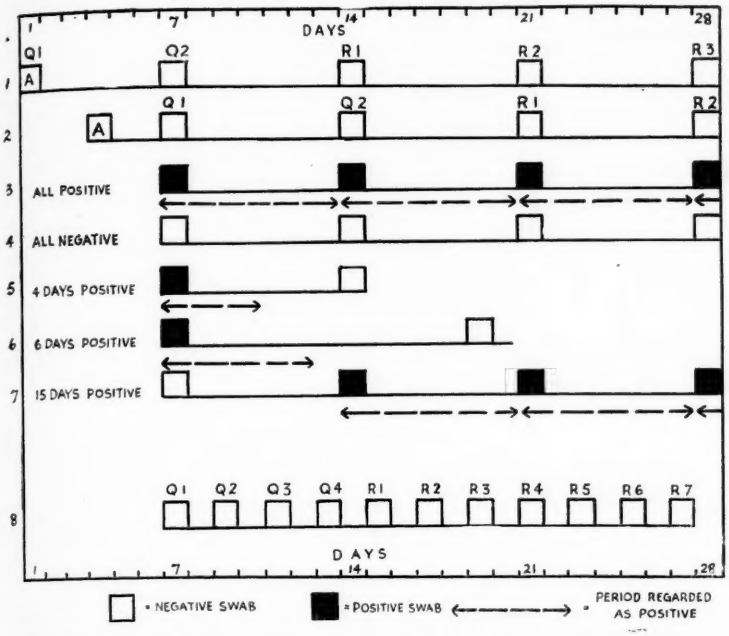

FIG. 365.—Hypothetical examples of the results of periodic swabbing, illustrating the definition of 'qualifying' swabs (1, 2, 8) and the method of calculating persistence of the carrier state in the inter-swab periods.

THE WARD ROUTINE AND ITS REORGANIZATION

During the control period, the dressing routine was, briefly, as follows: The nurse prepared the trolley, uncovered the wound, and then washed her hands. She next performed the wound toilet and rebandaged the wound, after which she proceeded directly to the next case without further washing of the hands. During the control period, too, there was a weekly ward round by the surgeon-in-charge, for which the wounds were prepared in advance by removal of dressings and covering with a sterile towel. This was done by a nurse, who uncovered all the wounds in turn, without intermediate washing of the hands or the use of a no-touch technique, and wrapped a sterile towel around them.

There were obviously many opportunities for the introduction of organisms into wounds at the time of maximum risk, i.e., when the wounds were exposed for dressing; the revised technique aimed at the reduction of the opportunities at this time. The revision was based on the recommendations in the *M.R.C. War Memorandum No.* 6 (1942), p. 5, and included the wearing of masks, the institution of a rigid no-touch technique, and the exposure of the wound for a minimum time. Instead of several nurses working singly, three nurses worked in a team as server, dresser, and runner. At the same time, the staff stopped the weekly round and arranged to inspect the wounds at the time of the routine dressing. This arrangement had the disadvantage that whereas, under the old system, the surgeon could see the 20 wounds in half to three-quarters of an hour, under the new he could only inspect 3–5 wounds in this time. The number was increased by having two wounds ready for him at a given time, but no modification can make this routine inspection as simple as that of the preliminary wrapping of the wounds for inspection in sterile towels. Nevertheless, it seems to us that the use of a loose temporary dressing greatly increases the risk of introducing organisms from the skin and bedclothes of the patient.

RESULTS

Load of Infection: Carriers.—The average carrier rates for *Staph. aureus* and *Str. pyogenes* in the patients and staff over the whole period are set out in *Table I.* The rates for the nursing staff and those derived from the weekly swabbing of the whole ward, which included patients who had been in the ward for several weeks, are sensibly higher than those from the patients swabbed on admission. It seems probable, therefore, that a small number of patients not carrying the organism at the time of admission tend to become carriers while in the hospital environment (cf. Wright, 1940; de Waal, 1941). *Table II* shows the average daily number of carriers of *Staph. aureus* and *Str. pyogenes* in the control and test periods, calculated by totalling the number of days of carriage shown by individual patients and dividing by the number of days in the period. The carrier rates indicate the magnitude of the upper respiratory tract sources of infection only if all carriers contribute a similar number of infective particles to the wound environment. The carrier state as determined by the swab depends not only upon the presence or absence

Table I.—CARRIER RATES FOR *Staph. aureus* and *Str. pyogenes* IN THE UPPER RESPIRATORY TRACTS OF PATIENTS AND WARD STAFF

PATIENTS AND STAFF	NO. OF PATIENTS AND STAFF	TOTAL NO. OF SWABS	PERCENTAGE OF POSITIVE SWABS	
			Staph. aureus	*Str. pyogenes*
Patients on admission	447	447	47·5	11·0
Patients during stay in ward	497	1219	56·7	15·8
Nursing staff	56	504	63·3	15·3

of the organisms, but also on the readiness with which they can be removed from the surfaces of the pharyngeal and the anterior nasal mucosa.

Table II.—Sources of Infection in Patients and Ward Staff

Sources of Infection	Control Period (70 Days)	Test Period (184 Days)
Upper Respiratory Tract (Patients) :—		
Staph. aureus, average daily number of carriers	15·0	13·2
Str. pyogenes, average daily number of carriers	2·4	4·3
Upper Respiratory Tract (Staff) :—		
Staph. aureus, average daily number of carriers	6·7	6·4
Str. pyogenes, average daily number of carriers	0·9	1·2
Wound (Patients) :—		
Staph. aureus, average daily number of carriers	15·2	13·1
Str. pyogenes, average daily number of carriers	6·7	6·5
Wounds (Staff) :—		
Staph. aureus, average daily number of carriers	0·2	0
Str. pyogenes, average daily number of carriers	0·2	0

The danger to a wound which the carrier represents, however, is dependent not so much on the carriage of the organism in itself as on the degree to which those organisms are liberated into the environment. The ease with which they may be liberated will be indicated partly by their presence on the swab and partly by the number of organisms isolated. An analysis of the results from nose and throat swabs cultivated in the two periods shows that the carriers were comparable in respect of the number of organisms removed by the routine swab. In most cases the growth of the indicator organisms was either conspicuous or profuse. The proportions of staphylococcal carriers who showed only a scanty growth in the control and test periods were 12·5 per cent and 11·1 per cent respectively. The corresponding figures for streptococcal carriers are 29·0 per cent and 28·5 per cent.

Table III shows the average number of patients in the ward on any one day, in three classes : (*a*) patients without wounds ; (*b*) patients with wounds, whose stay in the ward was so short that no more than the qualifying swabs could be taken, or patients with wounds which, being infected by both *Staph. aureus* and *Str. pyogenes* on admission, could not be used for the study of added infection ; and (*c*) patients with wounds suitable for the study of added infection.

It will be seen (*Table II*) that the average daily load of *Staph. aureus* was slightly greater during the control period ; that of *Str. pyogenes* greater during the test period. It is noteworthy that this is true both of the staphylococcal wound infections and carriers and of streptococcal carriers ; further, the similarity of the nasal staphylococcal carrier rate and the staphylococcal wound infection rate is striking, though its significance as an indication of any immediate relation between nasal carriage and wound infection is doubtful.

In the control period 17·1 per cent of all the wounds admitted to the ward were available for the study of added infection ; in the test period, 26·5 per cent. In the latter period, it will be remembered, a proportion of the patients was being treated with penicillin, and the figures in this table do not distinguish the treated from the untreated patients. If, from the figure for ' available wounds ', all those which were at any time treated with penicillin are excluded, the remainder form 17·5 per cent of all the wounds admitted. Penicillin treatment, by rendering some of the wounds sterile, has given us an increased number in which it would be possible to demonstrate added infection.

Load of Infection : Air and Dust Contamination.—Using a slit sampler (Bourdillon et al., 1941), we made a small survey of the bacterial flora of the ward air during the whole day. The slit of the sampler was about 5 ft. from the floor, and all the samples were taken on blood-agar plates which were incubated for one day anaerobically, followed by one day aerobically. Total counts for the various times of the day and night, taken on different days, are shown in *Fig.* 366. There was a large peak during the time of bedmaking and sweeping, but within one hour the count had fallen to a low level, which was maintained throughout the morning. Counts in the afternoon were much more variable, probably because there was a fair amount of movement of patients in this period.

Table III.—Distribution of Classes of Patient in the Ward

Classes of Patient	Control Period (70 Days)	Test Period (184 Days)
Number of patients admitted to the ward	146	320
Average daily number of patients in ward	25·4	29·4
Patients without wounds :		
Average daily number	3·4	6·9
Average stay in wards in days	6·7	12·0
Patients with wounds, but not included in the survey :		
Average daily number	14·3	12·5
Average stay in ward in days	10·9	14·6
Patients with wounds, included in the survey :		
Average daily number	7·7	10·0
Average stay in ward in days	28·2	32·4
Percentage of patients with wounds included in the survey, of the total number of wounded patients	17·1	26·5

During the hours from 9·0 a.m. to noon, when most of the dressing of wounds was done, 17 samples were taken in the ward on different days, with an average yield of 41 colonies per cu. ft. From all these 17 samples of 2 cu. ft., 5 colonies of *Str. pyogenes* were obtained. Fifteen samples taken in one morning, between 9·0 a.m. and noon, in the ward dressing station, yielded an average count of 67 per cu. ft., with a total of 3 colonies of *Str. pyogenes*. *Staph. aureus* was

even less common. At intervals during the same morning as these samples were taken in the dressing station, eight 9-cm. blood-agar plates were left open at bench height for 15 minutes each.

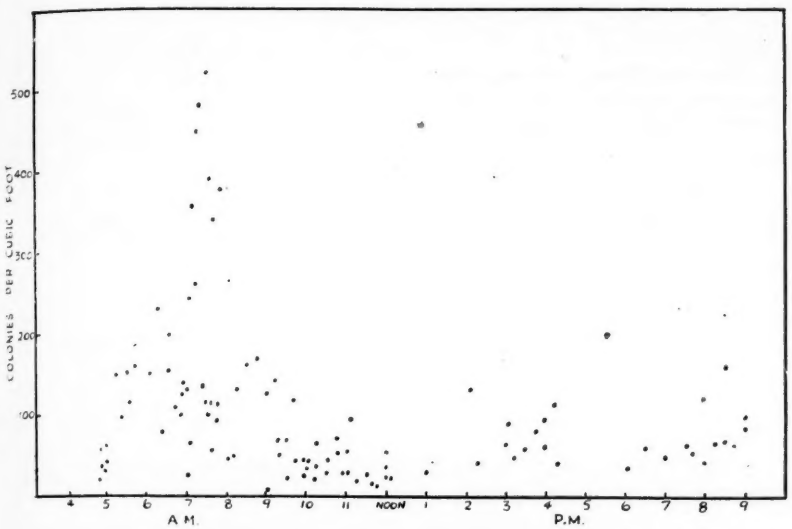

FIG. 366.—Composite diagram of bacterial contamination of ward air; 2 cu. ft. samples at different times of day.

The average number of colonies developing on these was 38, and one plate showed a colony of *Str. pyogenes*. Streptococcal contamination of the air was not, therefore, considerable. It is possible that, had the sampler been nearer the floor, at the level of a wound dressed in bed (i.e., about 30 inches), a greater degree of contamination would have been found, for dust gathered from under the bed of a patient with a streptococcal infection yielded approximately 1,000,000 colonies of *Str. pyogenes* and 400,000 colonies of *Staph. aureus* per gramme. Blood-agar plates exposed under the bed while it was being made, and under the curtains separating the beds, while they were being shaken, also showed several colonies of *Str. pyogenes*.

Incidence of Added Infection.—During the control period of 10 weeks, we observed 8 cases of added infection—5 streptococcal and 3 staphylococcal. In the 26 weeks of the test period there were 9 cases in all, 1 streptococcal and 8 staphylococcal. There was thus a reduction in the average weekly incidence from 0·8 to 0·3 cases. These crude rates do not, however, represent the whole picture, for the wounds were at risk for varying times. A truer picture is obtained by calculating the incidence of infection in wound-weeks at risk, as indicated above. *Table IV* gives the relevant data calculated in this way. The added infection rates are conspicuously lower in the test period, compared with the initial control period. The *Str. pyogenes* rate drops from 13·9 per cent to 0·75 per cent, and the *Staph. aureus* rate from 100 per cent (3/3 cases) to 17·8 per cent.

DISCUSSION

Added Infection.—The data presented in *Table II* show that there was no notable difference in the magnitude of the load of infection present in the ward during the two periods to be compared. The only striking difference in the conditions lies in the greater number of susceptible wounds during the test period, largely, it seems, due to the treatment of a proportion of the wounds with penicillin (*Table III*). With these facts established it seems justifiable to compare the incidence of added infection in the test and control periods (*Table IV*). In all groups of patients there was a marked reduction; for example, with the figures for added infection and re-infection with *Str. pyogenes*, the difference is 13·1 per cent ($SE_d = \pm 3{\cdot}5$, $d/SE_d = 3{\cdot}7$, $P = 0{\cdot}0002$). With added infections alone, the difference is 14·7 per cent ($SE_d = \pm 3{\cdot}6$, $d/SE_d = 4{\cdot}1$, $P = < 0{\cdot}0001$). These are differences which might have occurred by chance about twice in 10,000 trials. With staphylococci the reduction in the added infection rate, statistically considered, is highly significant ($d = 82{\cdot}2$ per cent, $SE_d = \pm 25{\cdot}1$ per cent, $d/SE_d = 3{\cdot}3$). The comparison is based on the supposition that added infection in each patient is an independent event. If the real incidence of staphylococcal added infection had been the same in both periods, an observed incidence of 3 in 3 cases in the control period might have been attributable to an immediate common cause. However, the 3 cases observed were well separated in time and space and can be considered as independent events.

The greater number of qualified wounds in the test period was due partly to its greater length, but also to the fact that more purely streptococcal infections were admitted, and that a number of the established staphylococcal infections were cleared up by treatment with penicillin, and so qualified for the observation of re-infection; indeed, of the 8 cases of added staphylococci in the test period, 6 were re-infections. In our view, re-infection constitutes less good evidence of added infection than the initial addition of bacteria to wounds that have never yielded these organisms, for it is reasonable to suppose that the probability of an *initial* contamination lying dormant for 10 days or more is much less than the probability of an *established* infection disappearing from the parts of the wound available for sampling and lying undetected for a

period in some closed pocket of the healing wound.

In sections 4–9 of *Table IV* patients treated with penicillin are considered separately from those not so treated, for it might be supposed that, during treatment with an efficient antibacterial agent such as penicillin, the risk of development of an added infection would be reduced. This was not so: half the cases of added and re-infection were observed in patients who were treated with penicillin. There was one case of an added staphylococcus during the course of treatment with the drug, as well as one case of re-infection by this organism. Three patients showed re-infection (one streptococcal and two staphylococcal) soon after the termination of treatment.

Table IV.—INCIDENCE OF ADDED INFECTION IN THE WOUNDS AT RISK

TYPE OF ADDED INFECTION	NO. OF TIMES ADDED SPECIES OBSERVED / NO. OF TIMES WHEN ADDITION MIGHT HAVE BEEN OBSERVED			
	Control Period (70 days)		*Test Period* (184 days)	
	No.	Per cent*	No.	Per cent*
All patients in ward :—				
1. Added species and reinfections :				
Str. pyogenes	5/36	13·9	1/134	0·75
Staph. aureus	3/3	(100)	8/45	17·8
2. Added species only :				
Str. pyogenes	5/34	14·7	0/103	0
Staph. aureus	2/2	(100)	2/22	9·1
3. Reinfections only:				
Str. pyogenes	0/2	(0)	1/31	3·2
Staph. aureus	1/1	(100)	6/23	26.0
Excluding patients who had penicillin treatment :—				
4. Added species and reinfections :				
Str. pyogenes			0/102	0
Staph. aureus			4/15	26·6
5. Added species only :				
Str. pyogenes			0/90	0
Staph aureus			1/9	11·1
6. Reinfections only :				
Str. pyogenes			0/12	0
Staph. aureus			3/6	(50)
Penicillin-treated patients :—				
7. Added species and reinfections :				
Str. pyogenes			1/32	3·2
Staph. aureus			4/30	13·3
8. Added species only :				
Str. pyogenes			0/13	0
Staph. aureus			1/13	7·7
9. Reinfections only :				
Str. pyogenes			1/19	5·3
Staph. aureus			3/17	17·6

* Percentages in brackets are based on less than 10 cases.

Coliform organisms were rare in the wounds studied, with the exception of those treated with penicillin. It is noteworthy that between Jan. 11 and Feb. 23 an organism of the paracolon group was isolated from six different wounds which were under treatment with penicillin and from one which was not so treated. Six of the cases showed this infection within one week, and, since we did not isolate this organism from any other wounds during the survey, it seems likely that we were observing a small epidemic of cross-infection. The organism was very active in destroying penicillin (Harper, 1943), a fact which may account for the apparent ease with which it colonized these wounds.

Limitations of the Method of Bacteriological Survey. — We might have been able to show even more striking differences between the added infection rates (*Table IV*) if we had had more patients available for study, particularly in the control period. The duration of this period was limited by circumstances outside our control, but the small number of patients available for the study was also in part dictated by the relatively long period (5–7 days) between consecutive swabs. For example, had we been able to sample every two days, the qualifying period of one week free from infection could have been determined from the results of 4 swabs taken over 8 days. In the case of patients whose ward-life was only 3 weeks, this would have meant that, after the qualifying period of 8 days, there would have been 7 occasions in 13 wound days (R1 – R7 in *Ex.* 8, *Fig.* 365) for the detection of added infection. As it was, about 7 days were available, in which only one swab was taken.

Some of the wounds yielded streptococci which could be assigned with certainty to one or other of Griffiths's types. In 9 of these wounds which, for a qualifying or longer period, had been consistently infected with one type, a new and serologically unrelated type appeared. There can be little doubt that cross-infection of streptococcal wounds with other streptococci occurred with some frequency, but the total number of wounds yielding typed streptococci was too small to make an estimate of this frequency. It is highly probable that, had a method of subtyping *Staph. aureus*, either serological (Cowan, 1939), or by bacteriophage filtrates (Fisk, 1942), been available, we should have been able to demonstrate similar cross-infection in the staphylococcal wounds.

McKissock, Wright, and Miles (1941) reported a reduction of added infection from 15·4 per cent to 1·1 per cent. Their rates are of added *Str. pyogenes* infections per number of patients admitted free from *Str. pyogenes*, whereas ours are rates of added infection per wound-weeks at risk, so that a strict comparison is not possible. The comparable figures from our data, i.e., infection rates per patient admitted free from *Staph. aureus* or *Str. pyogenes*, showing a newly

appearing organism on any swab after the admission swab, are 70·5 per cent and 23·2 per cent respectively in the control period, and 47·6 per cent and 9·3 per cent in the test period, the reduction being 23·9 per cent for *Staph. aureus* and 13·9 per cent for *Str. pyogenes* (*Table V*). But in any event the populations of wounds and patients studied are not comparable, for the proportion of open wounds admitted with established infection was much greater in our investigation. On the average there were 14·2 wounds infected with *Staph. aureus* and 6·6 with *Str. pyogenes* in the ward on any one day. Thus the large load of infection among the wounds and the small number remaining in which added infection could be demonstrated, greatly diminished the likelihood of a clean-cut diminution like that of McKissock et al.

Table V.—TOTAL ADDED INFECTION RATES (EXCLUDING RE-INFECTIONS).
(Considering all patients with negative admission swabs who had at least one subsequent swab at no matter what interval.)

	No. of Cases		Diff. (d)	SE_d	d/SE_d
	Control	Test			
Total free from *Str. pyogenes*	56	97			
Added *Str. pyogenes*	13	9			
Per cent added *Str. pyogenes*	23·2	9·3	13·9	5·9	2·36
Total free from *Staph. aureus*	14	42			
Added *Staph. aureus*	10	20			
Per cent added *Staph. aureus*	70·5	47·6	23·9	15·4	1·55

Nevertheless, the association of a drop in the wound-week infection rate with a revision of the dressing technique is sufficiently striking, and it confirms the conclusions of McKissock, Wright, and Miles that added infection is susceptible of reduction by such a revision. Of the more important changes in technique we may mention the introduction of a rigid no-touch technique, of masking the staff, of a routine wash of the hands after the completion of each dressing, the abolition of temporary dressing with sterile towels for convenience of inspection by the surgeon, and the co-ordination into a single team of three persons who previously had acted individually as separate dressers. It should be noted that the revision of the technique did not include any definite measures for the reduction of air-borne bacteria. It remains to be demonstrated that with small wounds exposed to the air for short periods during dressing the risk of air-borne infection is large enough to demand elaborate precautionary measures. Air-borne infection doubtless occurs, but the contact infections, both immediate and remote, constitute a much greater risk, and one which can be reduced by relatively simple revision of existing aseptic measures.

SUMMARY AND CONCLUSIONS

1. During a control observation period of 10 weeks, the added-infection rate per wound-week at risk in a surgical ward dealing mainly with septic wounds of the hands was 13·9 per cent for *Str. pyogenes* and 100 per cent for *Staph. aureus*.

2. During a test period of 26 weeks, after the adoption of a revised wound dressing technique, the added-infection rates were 0·75 per cent for *Str. pyogenes* and 17·8 per cent for *Staph. aureus*.

3. The carrier and infection rates for *Str. pyogenes* and *Staph. aureus* in the noses, throats, and wounds of the patients and in the noses and throats of the nursing staff were similar in both periods. The number of wound-weeks at risk was higher in the test than in the control period. The reduction in added infection may therefore with confidence be attributed to the revision of the dressing technique, which was made in accordance with the principles laid down in the *Medical Research Council War Memorandum No.* 6.

We are indebted to Mr. William Gissane, Clinical Director of the Birmingham Accident Hospital, for his advice and help; to Sister J. M. Taylor for her constant collaboration in the revision of the dressing techniques; and to Dr. Elizabeth Topley for her extensive work in sub-typing the streptococci isolated.

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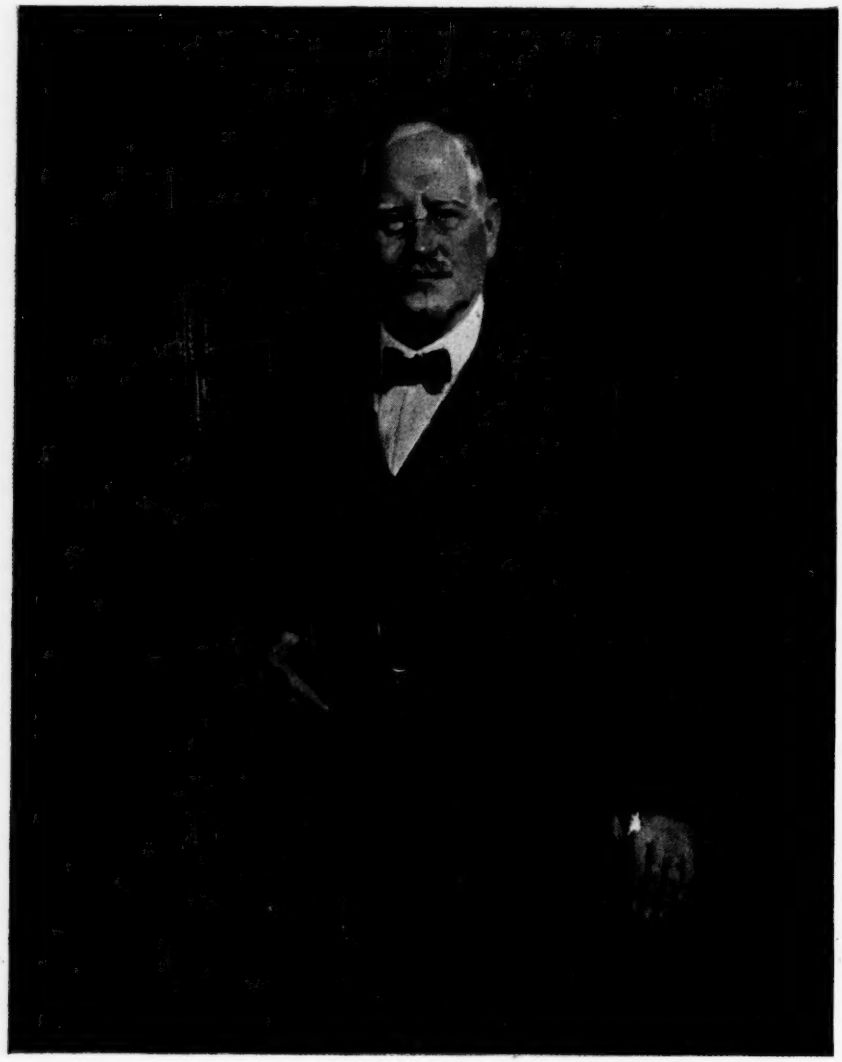
In Memoriam

ERNEST WILLIAM HEY GROVES

(1872—1944)

HEY GROVES was largely responsible for the foundation of the BRITISH JOURNAL OF SURGERY. He was its first Editor: he continued to edit it with tireless devotion until his retirement in 1941.



From the painting by Moussa Ayoub in the possession of Mrs. Hey Groves.

ERNEST WILLIAM HEY GROVES
Editor of the "British Journal of Surgery"
1913–1941

An appreciation of his work and service appeared in Number 114, Volume XXIX, of the JOURNAL.

Hey Groves earned and received many honours, but he built for himself his best monument in the BRITISH JOURNAL OF SURGERY, where his services are gladly and thankfully remembered.

SHORT NOTES OF RARE OR OBSCURE CASES

A CURIOUS CASE OF AIR EMBOLISM

By P. R. EVANS, Major, R.A.M.C. and R. S. MURLEY, Captain, R.A.M.C.

CASE REPORT

The patient in this unhappy case was an officer, aged 21, in an Armoured Regiment. While north of the river Sangro in Italy, at 09.00 hr. on Nov. 28, 1943, he was wounded by a shell fragment which entered the left side of his face. The regimental medical officer noted a hæmatoma of the cheek with free bleeding. He could locate no bleeding vessel, but managed to control the hæmorrhage with a skin suture and a firm dressing. The patient's pulse was weak and he was evacuated to an advanced dressing station where the suture was removed and the wound redressed. He arrived at the main dressing station in poor shape and severely shocked, was given 1080 c.c. of blood by a Field Transfusion Unit (F.T.U.), and evacuated to a Casualty Clearing Station (C.C.S.). Here he spent the next two nights and received a further 1080 c.c. of blood from a second F.T.U.

He was transferred to the Advanced Section of No. 1 Maxillo-facial Unit at another C.C.S. on Nov. 30. On admission, at 15.00 hr., he was in fair condition, T. 100·2°, P. 148, B.P. 140/80. At this time several instances had occurred of men developing severe malaria two or three days after wounding, and in view of this patient's fever, a blood-film was taken to exclude malaria. This film was negative and the spleen was not palpable.

There was a wound of the left infra-orbital region, approximately ½ in. in diameter and situated ½ in. to the left of the base of the ala nasi. The wound was crusted over with clot and there was moderate swelling of the cheek and left side of the neck, with bruising of the eyelids, subconjunctival hæmorrhage, and infra-orbital anæsthesia. He was completely blind in the left eye.

A radiograph showed a comminuted fracture of the left maxillo-malar compound and a large metallic foreign body in relation to the transverse processes of the atlas and axis on the left side.

The surgeon decided to undertake wound toilet. At 18.00 hr. a sister was about to inject alopon and scopolamine premedication. Immediately the needle pierced the skin the patient tried to climb off his stretcher and said " I want to go home." His temperature was taken again and found to be 102·6°. At 19.00 hrs. he was seen by the physician. The temperature was 104° and the patient was semi-conscious. Quinine dihydrochloride gr. 10 was injected intravenously. A second blood-film showed malignant tertian rings in fairly large numbers. Atebrin musonate g. 0·3 was given intramuscularly.

Next morning the patient was conscious, although not quite rational, and his speech was slurred and hesitant; T. 101·2°, P. 110. He was seen by Major E. C. Zorab, R.A.M.C., eye specialist of No. 2 Mobile Ophthalmic Unit, who found no perception of light in the left eye but clear media, a perfectly normal fundus, and a non-reacting pupil. He suggested that there was some interruption of the optic nerve due to hæmorrhage or compression by a bone splinter or, just possibly, due to a malarial lesion. An oral course of quinine, gr. 10 t.d.s., was started.

During the next two days the patient improved. The temperature oscillated between 100° and 102°, but had a downward trend and he felt better and was rational. On Dec. 3 he felt quite well when seen by the surgeon at 09.00 hr. The physician, arriving an hour later, found him feeling well. He complained of some sort of sensation in his left cheek, but his speech was still slurred and it was impossible to make out what he was saying. His breathing was rather 'bubbly.' He was stated by the patient in the adjoining bed to have been quite all right for the next ten minutes. Then he tried to cough up some sputum without much success. At 10.20 hr. he was unconscious and acutely dyspnœic. Ten minutes later he died, clear froth running out of his mouth terminally.

At Autopsy.—The missile had passed from the wound in the left cheek through the antrum and posterolateral antral wall, maxillary tuberosity, pterygoid laminæ, pterygoid muscles, and divisions of the mandibular nerve and internal maxillary artery. It had fractured the styloid process and torn the upper part of the internal jugular vein in its lateral aspect, finally lodging in the rectus capitis lateralis muscle of the left side. Bleeding must have been controlled only when the tension in the track became high because blood had tracked extensively among the structures of the left side of the neck. Possibly the blindness of the left eye was due to pressure of blood which had tracked up from the region of the pterygo-maxillary fissure, but no convincing evidence of this was found. The eye and orbit were dissected by Major Zorab, who found no abnormality apart from a large subconjunctival hæmorrhage. The riddle of the blind eye was unsolved.

Subcutaneous emphysema and distension of the superficial veins were noticeable on the upper part of the right anterior chest wall. There was emphysema of the anterior mediastinum and the lung roots, and less marked emphysema of the lower part of the neck. The lungs exhibited acute alveolar emphysema and extensive interstitial emphysema with venous congestion and œdema posteriorly. Large bubbles of blood, as well as small bubbles of air in œdema fluid, could easily be expressed from the cut surface of the lungs. Before the heart was opened the azygos vein was accidentally divided and air bubbles were seen to escape from it.

The outside of the heart appeared normal. The right auricle was opened before the venæ cavæ had been divided and was found to contain only bubbles of blood and no clot, as did also the right ventricle and the pulmonary artery and its divisions. The left auricle and ventricle contained post-mortem thrombus and blood without bubbles.

The spleen was enlarged and congested. A smear from it revealed moderate numbers of *Pl. falciparum* rings.

Comment.—This patient had a severe wound needing specialized treatment. He ran into three dangers in his passage to the appropriate medical unit. The first, hæmorrhage, was controlled by the regimental medical officer. The second, shock, was dealt with by two blood transfusions and staging in several units. He

then reached the Advanced Section of the specialist unit at a C.C.S. and encountered the third hazard, 'post-traumatic' malaria. This was controlled by immediate treatment with quinine and all seemed favourable for evacuation to the base section of the Maxillo-facial Unit for definitive treatment when he died, five days after being wounded, of a freak accident.

Autopsy prevented the death being written off as due to a complication of severe malaria and one is left to conjecture how this man did die.

The cheek wound was sealed with firm clot and there was no tear in the pharynx. Air must have entered the wound track via the nose and maxillary antrum. He had been warned not to blow his nose and was not seen by the other patient in the ward to do so. Presumably the clot softened and the air-track communicated with the tear in the internal jugular vein. He was sitting propped up in bed, the 'thoracic pump' produced a negative pressure in the vein and air was sucked in. This produced first a sensation in the cheek and later respiratory distress as the air filled the right heart and pulmonary arteries. Pulmonary œdema developed and aggravated the distress. Overbreathing into the reduced alveolar space stretched the alveoli and ruptured some of them, and air then escaped into the interstitial tissue and tracked into the mediastinum, up to the neck and over the chest wall.

Could anything have been done to save this patient? An operation which included discovering and treating the jugular vein tear would have been difficult, prolonged, and probably fatal, for as soon as he recovered from shock he developed severe malaria which was controlled but not cured when he died.

One must wonder, however, whether the pressure in the jugular vein would have been positive instead of negative, causing hæmorrhage instead of air aspiration, if he had been lying flat on the morning of Dec. 3, instead of being propped up in a sitting position because he was so much better and more comfortable that way.

SUMMARY

An officer received a facial wound involving the maxillary antrum and internal jugular vein. Having survived hæmorrhage, shock, and cerebral malaria, he died of air embolism on the sixth day.

DUODENAL DIVERTICULUM SECONDARY TO A GASTRIC ULCER

By R. MILNES WALKER

ROYAL HOSPITAL, WOLVERHAMPTON

Examples of ulcer diverticulum of the duodenum are not infrequent, and their mode of formation is quite clear; they are formed, as has been demonstrated by Edwards (1939), by the contraction of bands of scar tissue in the wall of the duodenum, and the diverticulum is the consequence of a bulge of the wall between such bands. Similar ulcer diverticula have been recorded in the stomach secondary to gastric ulcer. The present case is recorded as it demonstrates the odd occurrence of a diverticulum of the duodenum resulting from scarring associated with a large gastric ulcer which gave rise to scar tissue involving not only the pylorus but spreading across the wall of the duodenum to a sufficient extent to bring about the circumstances suitable for the development of a diverticulum. Mr. Harold Edwards tells me that he has not previously come across this state of affairs, but that he has met with an example of the reverse, viz., a gastric diverticulum secondary to a duodenal ulcer.

CASE REPORT

Alfred B., aged 51, was admitted to The Royal Hospital, Wolverhampton, under the care of Dr. J. H. Sheldon, on Dec. 18, 1941; he gave a history of gastric symptoms for the previous fifteen years, and medical treatment had never given more than temporary relief. Radiographic examination in 1933 had shown an excavating ulcer on the lesser curvature, while at a recent examination he was reported as

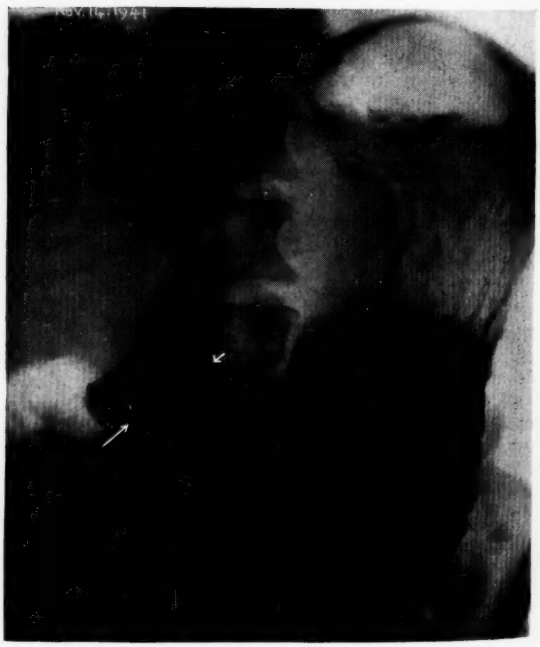


Fig. 367.—Radiograph after a barium meal showing the duodenal diverticulum.

having a dilated stomach with considerable four-hour residue; subsequent examination of the plates (*Fig.* 367) showed the presence of the diverticulum, but this had not been recognized before the operation. No tumour was palpable.

A partial gastrectomy of the Polya type was undertaken on Dec. 23; a gastric ulcer involving the lesser curvature and posterior surface was dissected off the pancreas leaving the base of the crater on the pancreas; the presence of a large diverticulum on the inferior margin of the first part of the duodenum was noticed as soon as that organ was examined, and care was taken to include this in the portion resected.

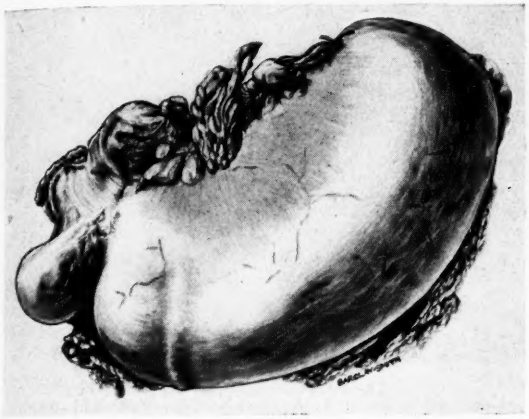


FIG. 368.—The resected portion of stomach from the anterior aspect.

The patient made a satisfactory recovery, and when seen 2 years and 2 months after the operation stated that he was in the best of health, and had had no gastric symptoms since his operation.

The specimen (*Figs.* 368, 369) consists of the lower half of the stomach, the pylorus, and about 3 cm. of the duodenum. On the lesser curvature of the stomach is a saddle-shaped ulcer, 4·5 cm. × 3·2 cm., extending rather farther across the posterior surface than the anterior; the posterior part is represented by a defect in the whole thickness of the wall of the stomach, 2·5 cm. × 1·9 cm., where the pancreas had formed the base of the ulcer. The pyloric opening is reduced to a firm ring, with an aperture only 1·4 cm. × 0·4 cm. Immediately distal to the pylorus, on the lower margin of the duodenum is a diverticulum 2·5 cm. deep, and 1·5 cm. wide at its neck; the widest diameter is slightly greater than that of the neck.

There is no sign of any scarring of the mucous membrane of the duodenum, and the lines of scar tissue seen on the external surface and corresponding folds of mucous membrane, which define the margins of the neck of the diverticulum, radiate from the gastric ulcer and extend right across the pylorus.

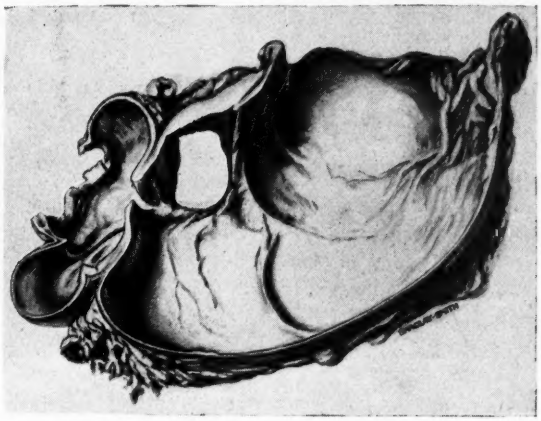


FIG. 369.—The same specimen laid open. Most of the floor of the crater of the ulcer was formed by the pancreas, and is represented by the hole in the posterior wall of the specimen. The pylorus is reduced to a narrow slit. The duodenum has been cut across 1 cm. distal to the diverticulum.

It can be assumed, therefore, that in the absence of any evidence of an active or healed duodenal ulcer, this unusually large duodenal diverticulum is the sequel to a chronic gastric ulcer.

I am indebted to Miss Barclay-Smith for the clear drawings of the stomach.

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A CASE OF RUPTURE OF A CONGENITAL DISCOID CARTILAGE

BY J. R. BELCHER, FLIGHT-LIEUTENANT, R.A.F.V.R.

THE following case seems worth reporting, as it was possible, pre-operatively, to demonstrate a tear in a congenital discoid cartilage by radiological means, following the technique recently described in this JOURNAL by Cullen and Chance (1943).

CASE HISTORY

HISTORY.—An airman, aged 19, reported sick with a painful left knee following a severe twist, the force being that of internal rotation of the tibia on the femur, sustained a few days before. On questioning, he stated that he had had an abnormal left knee, which gave a loud click on flexion after full extension, as long as he could remember, but which had given little trouble apart from occasional pain after prolonged exercise, and which had sustained no trauma that could account for this abnormality.

Since the recent accident the knee had been painful and swollen, and he had been unable to run and walked with a limp. When first seen a diagnosis of a congenitally abnormal cartilage was made, and symptoms were thought to be due to an aggravation

of his normal ache after exercise. However, it was not until fourteen days later, when the pain and effusion showed no signs of receding despite treatment, that the possibility of a tear was considered.

ON EXAMINATION.—The quadriceps showed little or no wasting. There was a mild degree of synovial effusion. Tenderness was present over the anterolateral compartment and posterior aspect of the joint, and there was pain on internal rotation.

FIG. 370.—Showing the normal medial meniscus.

On flexion after full extension there was a loud click when the knee was at approximately 90°, the tibia shook on the femur, and the cartilage could easily be felt coming outwards just in front of the collateral ligament.

Arthrography was performed, following the technique already mentioned, but only about 50 c.c. of air could be introduced without producing a marked degree of tension; the whole joint was firmly

FIG. 372.—Showing the upper surface of the meniscus. The arrow marks the site of the upper end of the tear.

bandaged from above downwards to try and ensure visualization of the cartilage. The medial side of the joint showed no abnormality (*Fig.* 370).

The lateral side showed a picture which we felt justified our making a diagnosis of a tear of a congenital discoid cartilage (*Fig.* 371).

A line of cleavage (approximately in the centre) could be seen with the major portion of the cartilage extending right across the joint and being apparently detached at its medial border.

In view of this and the fact that there had been considerable disability since the recent accident, it was decided to remove the damaged cartilage.

AT OPERATION.—The cartilage was removed through an anterolateral incision; some difficulty was encountered in detaching it from the centre of the joint, particularly at its anterior end. *Figs.* 372, 373 show its appearance after removal.

As can be seen, it was a congenital discoid cartilage with a well-marked tear on its inferior surface which extended up and outwards, but which did not extend as far as the upper surface; just behind the tear there was a niche where the cartilage was not attached to the centre of the joint and which no doubt represented

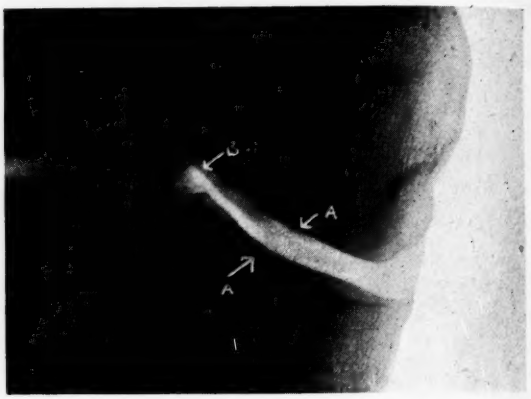


FIG. 371.—Showing the abnormal side. A, A, indicates the line of the tear; B, the shadow of the inner and apparently detached part of the disc.

an attempt at the normal disappearance of the central part of the disc. The lesion is in fact just as was anticipated from the radiograph, the upper and larger shadow representing the main body of the disc and

FIG. 373.—Showing the quadrilateral detached fragment, with the arrow marking its posterior end.

the smaller one the fragment detached from the lower surface. It shows the actual line of cleavage more clearly than the photographs.

PROGRESS.—The patient made an uninterrupted recovery. The rate at which the tone and power of his quadriceps returned was very good and he was up and about six days after the operation. He was discharged on the twelfth day with flexion 10° short of normal, full extension, but still a little wasting of the quadriceps.

COMMENT

Although it was possible to make the diagnosis on clinical grounds with a fair degree of certainty,

the radiograph is of more than academic interest, as it demonstrates the possibilities of the technique in cases where the diagnosis is more doubtful. The reaction in each of the cases so far done has been very slight. The air is rapidly absorbed and the effusion minimal. It seems a justifiable procedure where the diagnosis on clinical grounds is at all open to doubt.

My thanks are due to S/L K. D. F. Morle for his excellent radiographs.

SUMMARY

1. A case of rupture of a discoid cartilage is presented.

2. Radiographs after injection of air into the joint are shown in which pre-operative diagnosis was possible.

REFERENCE

CULLEN, C. H., and CHANCE, G. Q. (1943), *Brit. J. Surg.*, **30**, 241.

REVIEWS AND NOTICES OF BOOKS

Recent Advances in Anæsthesia and Analgesia (including Oxygen Therapy). By C. LANGTON HEWER, M.B., B.S. (Lond.), D.A. (Eng.), Senior Anæsthetist, St. Bartholomew's Hospital and St. Andrews' Hospital, Dollis Hill; etc. Fifth edition. 8 × 5¼ in. Pp. 343 + viii, with 141 illustrations. 1944. London: J. & A. Churchill Ltd. 18s. net.

IT is but a year since the fourth edition of this invaluable book was published, and yet this fresh edition contains a wealth of new material.

The arrangement of the chapters remains the same, and the new work has been introduced with the author's customary skill. An additional chapter on Anæsthetic Charts and Records has been placed at the end of the book and is of great interest to all anæsthetists.

The new work gives additional information on anæsthesia for thymectomy, general analgesia with ethyl chloride and trichlorethylene, intravenous general analgesia with procaine, improved apparatus for controlled respiration, pethidine analgesia in obstetrics, pressure infiltrators, fractional caudal block, and suction for oxygen cylinders.

There are 141 illustrations, of which 21 are new. These give definite assistance to the reader in assimilating such a vast store of knowledge.

This book has become indispensable to specialist anæsthetists, and both author and publishers are to be congratulated on their successful endeavour in presenting us once again with *Recent Advances in Anæsthesia and Analgesia*, in which we find in very fact 'recent advances'.

The Surgery of Abdominal Trauma. By GEOFFREY E. PARKER, M.B., B.Ch., F.R.C.S. (Eng.), Surgeon to the French Hospital, London; Surgeon to the Woolwich War Memorial Hospital; Surgeon to the Erith and District Hospital, Kent; Major, R.A.M.C. With a Foreword by Col. J. M. WEDDELL, C.B.E., F.R.C.S. 8 × 5⅛ in. Pp. 120 + viii, with 10 illustrations. 1944. London: J. & A. Churchill Ltd. 10s. 6d. net.

THE author was Surgeon to the 24th Field Surgical Unit in the forward areas of North Africa and Italy and he dedicates his book to the 46th Division, from which most of his cases were drawn. As Col. Weddell says in his Foreword, "A recovery-rate of 66 per cent is a most creditable record". This in no small measure must be due not only to the author, but to the anæsthetist, Capt. J. B. Wyman R.A.M.C., and the team-work of nurses and orderlies of the Field Surgical Unit. As the author also states in his Preface, the smooth running of a Field Surgical Unit would not be possible but for the assistance of many Senior Officers, especially the A.D.M.S. and Field Ambulance Commanders, by whom he was relieved of much administrative and military work, which all surgeons agree should be the case.

The book consists of case records of 94 cases of abdominal trauma. "The notes of the cases were written at the time of the receipt of the casualty and on completion of the operative treatment in the theatre". The 'follow up', which necessarily must be incomplete, "has been done with the greatest care, and in Italy, with short lines of communication, it was possible for the Forward Surgeons to get down to Base Hospitals".

It is to be hoped that opportunity will later be given to the author to complete the follow-up. The number of cases, though small, will, however, make a much more valuable record and addition to our knowledge, if fully completed, than a much larger though incompletely recorded series.

It has been cynically said that next to discussing and describing your own successes, it gives to some the greatest pleasure to discuss the other fellow's failures. The curse of many sets of surgical statistics is 'the successful series'. All the cases are wanted —good and bad—those in which an error of judgement has been perpetrated, as well as those in which the diagnosis was marvellous and the executive technique brilliant.

The author of this book has given us honestly and straightforwardly a consecutive series. Errors of commission and omission are not glossed over but stressed, and he should be thanked for placing his knowledge and experience at the service of others.

"Experience is the Child of Thought and Thought is the Child of Action. One cannot learn men from books", and "What is all knowledge, too, but recorded experience". The author must have learned much from the fourteen abdomens he opened unnecessarily on a mistaken diagnosis.

He naturally begins with the diagnosis of an abdominal injury. The severe one offers few difficulties, but he warns us to beware of: (1) Penetrating injuries in the lower thoracic region which may or may not have entered the abdomen; (2) Abdominal parietal wounds in which intra-abdominal lesions occur as the effect of blast; (3) Buttock wounds which may have penetrated the pelvic peritoneum.

Auscultation of an acute abdomen he considers of great importance, but is sure that the presence of

free blood, unlike other fluids, does not produce "a *silent* abdomen".

He condemns any attempt to elicit shifting dullness; movement of the patient only makes shock worse and provokes hæmorrhage.

Vomiting is usually absent as it only causes more pain and the patient tries to prevent it.

He naturally and properly insists that all cases should be catheterized to help the diagnosis, and operative approach and proctoscopic examination should never be omitted in lower abdominal wounds.

Absence of liver dullness does not carry much weight with him, nor does a pre-operative X-ray examination. "It is the damage they have done and not the fragments themselves which threaten the patient's life".

In cases of great difficulty a pre-operative radiograph showing free gas below the diaphragm might be the determining factor.

He supports the dictum of the late Sir Cuthbert Wallace, viz., to look and see rather than wait and see. The time factor is one of the surgeon's greatest allies.

Like all surgeons who have dealt with abdominal wounds, he prefers to wait until the patient is "warmed up" but not to waste invaluable time in resuscitation; as in the treatment of ruptured peptic ulcer in civil life, secondary shock and serious complications are prevented by quick operation. For this reason it is not clear why neither blood nor plasma "should be given at any point on the line of evacuation where surgery is not immediately available except in those rare cases where it is virtually certain that hæmorrhage is not taking place and the problem is one of uncomplicated shock". The time for operation was always decided by consultation between the surgeon and anæsthetist. The premedication used was omnopon (gr. $\frac{1}{3}$), scopolamine (gr. $\frac{1}{150}$) one hour before, and atropine (gr. $\frac{1}{100}$) half an hour before, operation. The former might in very urgent cases be given intravenously five minutes before induction of the anæsthetic, which was C_2E_3 mixture. The Oxford Vaporizer was also found most useful.

The midline incision was found to be the best whatever the position of the wound. The operative space can always be increased by suitable right-angled extensions to deal with liver, spleen, or kidney lesions.

The entry and exit wounds were always thoroughly inspected from within when the abdomen was opened, any necessary enlargement being made. They were excised after the abdomen was closed. This routine was followed even if the entry was through the buttock; if hæmorrhage is going on it can be temporarily controlled by packing and dealt with later. Naturally intraperitoneal hæmorrhage must first be dealt with.

Sulphadiazine emulsion was freely used for coating suture lines, etc. If packs had to be left in to control hæmorrhage, then it was found that daily moistening with iodine, 1 part, glycerin, 9 parts, made subsequent removal easier.

The peritoneum and fascia were always closed with continuous catgut, a few tension catgut sutures were put into the fascia, but no deep through-and-through skin stitches, the latter took up only the subcutaneous fat. The subfascial planes were always drained by a slip of corrugated rubber. There was only one 'burst abdomen' in the ninety-four cases, and that incision had been closed without drainage.

The technique for the repair of viscera became to some extent standardized, splenectomy for all splenic wounds, nephrectomy for disorganized kidney and injury to pedicle, but some kidney wounds were sutured. Gunshot wounds of the kidney may be much less painful than kidney injuries in civil life, because in the latter the renal fascia and capsule are not usually ruptured. A word of warning is given urging that the ureter and pelvis of the kidney should always be dissected from the renal pedicle before ligaturing the latter. Risk of recurrent hæmorrhage from the renal artery is thus diminished.

Wounds of the liver, as the result of experience, should be sutured and packed; those on the posterior surface can be explored through a posterior transthoracic incision.

Only one in five cases required resection of bowel, the indications for which are: (1) Gross destruction of a length of intestine. (2) Damaged mesentery near to the bowel wall depriving the bowel of its blood-supply. (3) Damaged main junctional vessels in mesentery remote from bowel. (4) Burned bowel. This must be looked for especially if there is no wound of exit and is more common than usually thought to be.

End-to-end anastomosis was used, as it is performed quickly; if there are many perforations a loop of bowel may be marsupialized and dealt with later.

A valvular cæcostomy is usually an essential in colon injuries, but often a colostomy is better. The latter naturally is definitely indicated in rectum injuries and should always be complete.

For bladder injuries a suprapubic cystostomy is better than the indwelling catheter and should be maintained for eight days at least.

The term 'retroperitoneal syndrome' is introduced as a diagnosis in some types of abdominal injury and accredited to Lt.-Col. Jolly. It seems a pity to introduce what may well become a cliché to describe retroperitoneal and pericolic hæmorrhage. The symptoms are all well known and have been recognized for many years by surgeons dealing with 'heavy-industry' and 'run-over' accidents. It is true that these cases cause great anxiety and difficulty to the surgeon, but it is better to be sure than sorry. There is a tendency to dismiss a doubtful case as 'extraperitoneal syndrome' and leave it at that. The condition may also be due to mesenteric thrombosis with no hæmorrhage.

The author must owe a special debt of gratitude to those who carried out the post-operative treatment. In spite of routine duodenal suction, transfusion, free use of chemotherapy, it is the nursing and encouragement given to the patient which plays such an important part.

It will repay any surgeon to study the case records carefully, and to experience the thrill which evidently the author got when he watched the recovery of some of them and also share his disappointments. The outcome of such a series must have been a cause of great satisfaction to the team, all of whom are to be congratulated on being physically fit to carry out such work under such conditions. They must all be:

"By nature honest, by experience wise,
Healthy by temperance and by exercise."

Vascular Responses in the Extremities of Man in Health and Disease. By DAVID I. ABRAMSON, M.D., F.A.C.P. 9 × 6 in. Pp. 412 + x, with 59 illustrations. 1944. Chicago, Ill.: The University of Chicago Press (London: Cambridge University Press). 30s. net.

THIS volume is the outcome of ten years' study of the literature of the subject, combined with the author's own experience of the many methods he

describes for investigating the peripheral circulation in man. Its particular virtue lies in the detailed account given of the venous occlusion plethysmographic method of estimating blood-flow, which, in spite of many theoretical criticisms, is regarded as the most satisfactory quantitative method at present available. No doubt it would be easier to apply in practice than it seems to be in the written description—for however clear the directions may be, demonstration must be clearer—and it is not fair to judge the method without a trial. But one gains the impression that attention to the many factors which have to be controlled if results are to be reliable makes it a method of physiological and clinical research, rather than a test which can be usefully employed as a routine measure for assessing the peripheral circulation in hospital patients.

Whether this be so or not, the book is of much value as a stimulus to greater accuracy in the examination and recording of peripheral vascular injury and disease, and it will amply repay study by any clinicians interested in this subject. In addition to the physiological control of the circulation, consideration is given to abnormalities produced by disease, to vasodilator and vasoconstrictor drugs, and to the effects of various therapeutic measures, such as sympathectomy. The bibliography shows clearly how much study, thought, and industry have gone to the making of the book.

The Rehabilitation of the Injured. Occupational Therapy. By JOHN H. C. COLSON, Technical Director of Rehabilitation, Accident Service, Royal Sheffield Infirmary and Hospital. With a Foreword by E. A. NICOLL, M.D., B.Ch., F.R.C.S. (Edin.). 8½ × 5½ in. Pp. 226 + xvi, with 196 illustrations. 1944. London: Cassell & Co. Ltd. 15s. net.

THE literature of rehabilitation is of two kinds: the generalizations and vapourings of the platform experts; and the much more rare—and welcome—utterances of those who know how to do the job and have the gift of imparting their knowledge to others.

The author of this book is among the latter, and his special field is occupational therapy. Most of this work is technical, and the reviewer is not in a position to pass judgement on the apparatus, materials, and methods of construction that Mr. Colston describes with a wealth of detail and illustration. However, if the technical sections are as sound as the parts of the book that a comparatively unenlightened surgeon can understand—those that deal with the care of the injured patient—then this is a work that can be recommended without a moment's hesitation.

Textbook of Surgical Treatment, including Operative Surgery. Edited by C. F. W. ILLINGWORTH, M.D., Ch.M., F.R.C.S.E., Regius Professor of Surgery, University of Glasgow. Second edition. 9⅝ × 6⅛ in. Pp. 564 + xii, with 230 illustrations. 1944. Edinburgh: E. & S. Livingstone Ltd. 30s. net.

IN its second edition this book is slightly increased in size and price and considerably improved by a new chapter on the treatment of burns and an addition of nearly 100 illustrations. It presents a series of accounts of the treatment of various surgical conditions written by a number of contributors. Each chapter is complete in itself and forms a useful guide for the student or surgical trainee in the principles of treatment of the condition discussed; there is in most cases a deliberate omission of such technical details as the operating surgeon would require. Whilst many of the accounts are excellent, there are still some items which we feel should be included in a work of this character, and to which we drew attention in our review of the first edition. Among more recent developments there is no mention of synthetic œstrogens or of castration in the treatment of carcinoma of the prostate. The statement that perineal hypospadias is "unsuitable for treatment" is neither true nor helpful; surely this is the type most in need of treatment.

The chapter on the surgery of the autonomic nervous system remains one of the best in the book, and the new chapter on burns gives good detailed instructions of the methods which have proved most satisfactory in the author's hands.

Pye's Surgical Handicraft. Edited by HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London; etc. Fourteenth edition, fully revised. 9 × 6 in. Pp. 628 + xi, with 724 illustrations. 1944. Bristol: John Wright & Sons Ltd. 25s. net.

THE issue of the fourteenth edition in its diamond jubilee year is a fitting tribute to the continued popularity of "Pye"; in itself this edition shows several changes when compared with its predecessors, and its utility is enhanced by much new matter which brings it right up to date. Mr. Hamilton Bailey has enrolled such a large number of contributors that the book is now a complete guide to all the duties of the house surgeon. It is arranged for easy reference, and the number of illustrations has been increased; some of the new coloured photographs are rather small, but are adequate for their purpose.

We are surprised to find no mention of the indigo-carmine test of renal function; it is more widely used in this country than the phenol-sulphonephthalein test which is described. In other respects this chapter is excellent.

The book is one of the most useful which the surgical dresser can possess and is almost an essential for the house surgeon; the new edition can be confidently recommended as a worthy advance on its forerunners.

Aids to Theatre Technique. (Nurses' Aid Series.) By MARJORIE HOUGHTON, S.R.N., S.C.M., D.N., Sister Tutor, University College Hospital; and MARGARET HARDING, S.R.C.N., S.R.N., S.C.M., Senior Theatre Sister, University College Hospital. With a Foreword by WALPOLE LEWIN, M.S. (Lond.), F.R.C.S., Capt. R.A.M.C., late Harker Smith Registrar and Assistant to Surgical Unit, University College Hospital. 6⅝ × 4⅛ in. Pp. 262 + xiii, with over 100 illustrations. 1944. London: Baillière, Tindall & Cox. 4s.

THIS is one of the *Nurses' Aid Series* which the publishers hope, when completed, will cover "the field of knowledge required from the modern nurse".

From the titles of the volumes already published, it appears that the education for examinations of the student nurse, like that of the medical student, is being directed more and more to the importance and prestige of the science; while the vocation and art of her profession is being submerged beneath a flood

of factual knowledge. In spite of what matrons and sister tutors may think and say, a nurse's profession is surely one to which a woman still may be born, and the nurse whom the examiners reject be a real loss to the community.

The publishers give no indication as to whether they propose to issue an "Aid to the Vocation of Nursing" or "Aids to Matrons, Home Sisters, and Sister Tutors" as a grand finale to the series, "illustrated where necessary".

As is stated in the Foreword, it is impossible to standardize operative procedures by different surgeons (thank goodness !), and herein is one of the theatre nurses' great difficulties. It should be possible, however, to agree on certain basic principles, some of which are not mentioned in the text, e.g., the ward nurse should see that the ward blankets are not taken into the theatre, but replaced by clean bath towelling. This is more important for a successful outcome of the operation than notes, X-ray films, etc., nor is a sick bowl out of place as an accessory, and the warm gown and blanket should be ready to cover the patient when he leaves the theatre rather than "when he returns to the ward".

There is no margin of safety for sterilization of theatre linen, yet the authors seem satisfied that "once a month drum tests are made to ensure that sterilization is efficient". On the other 27 days it may be inefficient, yet chemical tell-tales could be inserted every day and any error in sterilization be detected.

In the chapter on anæsthetics there is no mention that the close liaison with the anæsthetist, who should have examined the patient pre-operatively, is more important than that "between the theatre and the ward". This liaison is all the more necessary as there are many more bad anæsthetists than anæsthetics. It is a pity that the Oxford Vaporizer is not mentioned.

The main part of the book consists of excellent photographs of instruments for various operations, with explanatory captions, all of which will be of great use to the theatre nurse in the course of her training and after.

Occasionally there is a running commentary which will whet the appetite of any surgeon, e.g., under the heading of abdomino-perineal excision of the rectum it is stated, "Both steps of the operation may be performed simultaneously by two teams". This must be staged only for very special occasions to prevent gate-crashing.

The nurse should be warned that when diathermy is being used all inflammable fluids should be removed from the vicinity of the apparatus. The ophthalmic surgeons should be supplied with local anæsthetics, as also should be done when the instruments are put out—the authors prefer "lay up"—for Rammstedt's operation or thoracotomy.

It is surely a mistake to say that Crutchfield's skull callipers "are inserted *through* the parietal bones" in the treatment of fracture or dislocation of the cervical spine. If the illustration on page 175 is taken during life, then the operator's technique appears to have run away with his reason. The technique of neurosurgery is exceedingly difficult and tedious, but well it may be, if all the instruments illustrated on page 187 are required for ventriculography.

There are a few avoidable misprints, that on the last line but one on page 215, should amuse Sir Harold, and Bonney-Reverain, on page 244, is at first sight almost the truth.

The book, however, does supply a real want amongst nurses. This first edition bears evidence of haste. It will, one is sure, be absent from subsequent editions, which will probably also incorporate many suggestions from theatre sisters all over the Empire.

But however many aids to knowledge may be published, let it always be remembered by the nurse that:

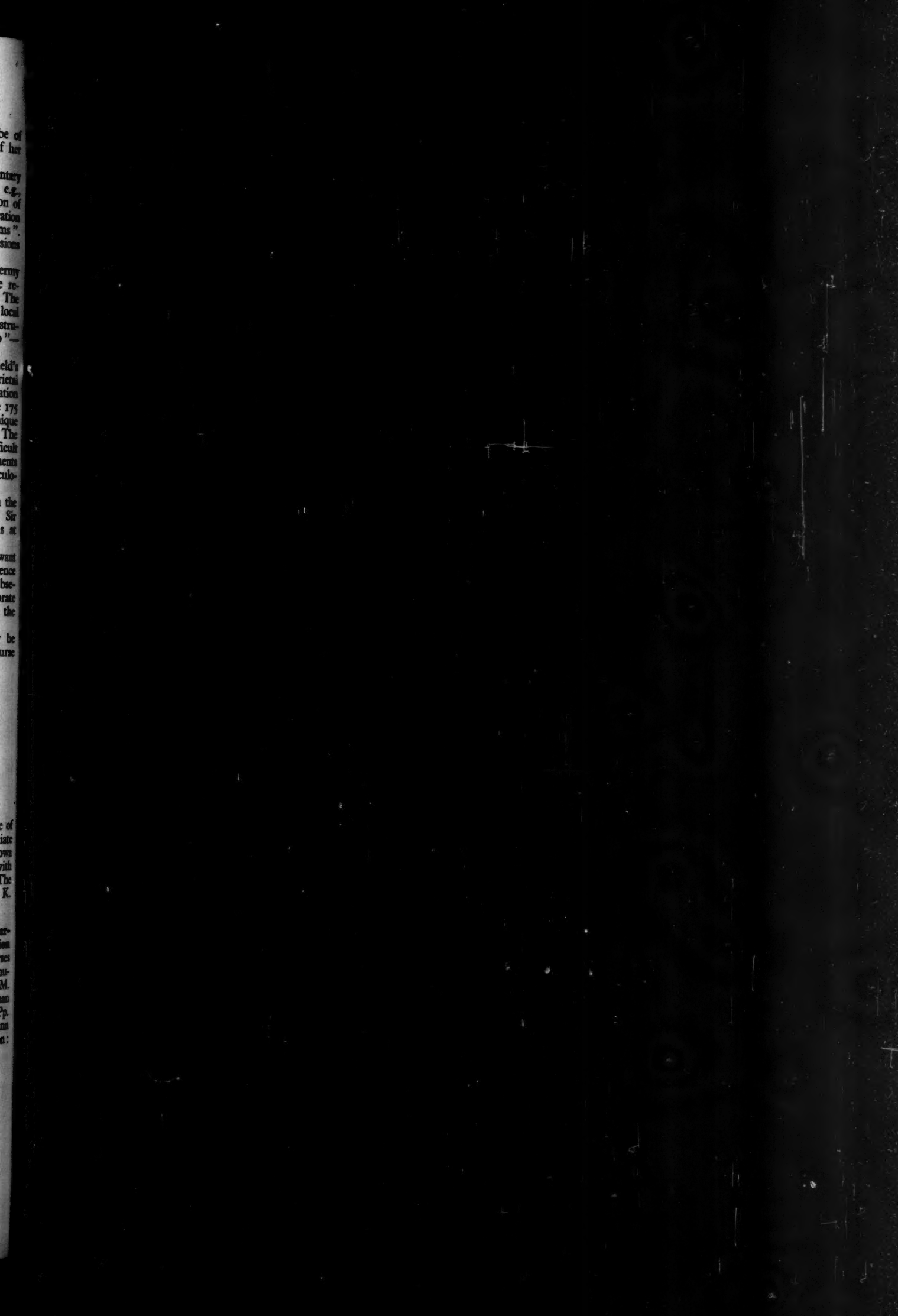
"Knowledge comes, but wisdom lingers."

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

Textbook of Anæsthetics. By R. J. MINNITT, M.D. (Liverp.), D.A., Lecturer in Anæsthesia, University of Liverpool; etc.; and JOHN GILLIES, *M.C.*, M.B., Ch.B. (Edin.), D.A., Consultant in Anæsthetics, Department of Health for Scotland; Anæsthetist, Professorial Surgical Unit, Royal Infirmary, Edinburgh; etc. Sixth edition. 8½ × 5½ in. Pp. 487 + viii, with 199 illustrations. 1944. Edinburgh: E. & S. Livingstone, Ltd. 25s. net.

Operations of General Surgery. By THOMAS G. ORR, M.D., Professor of Surgery, University of Kansas School of Medicine, Kansas City, Kansas. 10¼ × 7¼ in. Pp. 723 + viii, with 1396 illustrations on 570 figures. 1944. Philadelphia and London: W. B. Saunders Co. Ltd. 60s. net.

The Urinary Tract. A Handbook of Roentgen Diagnosis. By H. DABNEY KERR, M.D., Professor of Radiology, State University of Iowa College of Medicine, and CARL L. GILLIES, M.D., Associate Professor of Radiology, State University of Iowa College of Medicine. 8 × 5½ in. Pp. 320, with numerous illustrations. 1944. Chicago: The Year Book Publishers Inc. (London: H. K. Lewis & Co. Ltd.) 34s. net.

The American Academy of Orthopædic Surgeons Presents Lectures on Reconstruction Surgery. Selected from the Instructional Courses of the Twelfth Annual Assembly, Chicago, January 23–24, 1944. Edited by JAMES E. M. THOMSON, M.D., Lincoln, Nebraska, Chairman of the Instructional Section. 10⅞ × 8¼ in. Pp. 568 + iv, with many illustrations. 1944. Ann Arbor, Mich.: Edwards Bros. Inc. (London: H. K. Lewis & Co. Ltd.) £2 9s. net.